610.5
J615
N45

JULY 1941

Volume II. Number 7.

DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

PUBLISHED MONTHLY for the PHYSICIAN in the GENERAL PRACTICE of MEDICINE

CONTENTS:

THE RECOGNITION OF MALADJUSTED PERSONALITIES BY DRAFT BOARDS (Page 212)
Hamilton Ford, M.D., Galveston, Texas

THE PSYCHOLOGIC AND ECONOMIC INFLUENCE OF ALCOHOL (Page 218)
H. Mason Smith, M.D., Tampa, Florida

RESUME OF METRAZOL—SHOCK THERAPY IN SCHIZOPHRENIA (Page 222)
Serge Androp, M.D., and Ethel Green, M.D., Catonsville, Maryland

NEUROLOGICAL COMPLICATIONS OF SULFANILAMIDE AND SULFANILAMIDE COMPOUNDS (Page 229)
Harrison Evan, M.D., Columbus, Ohio and Ferdinand Welebir, M.D., Los Angeles, California

THE INCIDENCE OF CONTRIBUTORY FACTORS AND ASSOCIATED CONDITIONS IN VASCULAR ENCEPHALOPATHY (Page 234)
Cullen Ward Irish, M.D., Los Angeles, California

EDITORIAL, SANITARIA

ASSOCIATE EDITORS

Peter Bassoe, M.D. ..... Chicago, Illinois
A. E. Bennett, M.D. ..... Omaha, Nebraska
Franklin G. Ebaugh, M.D. ..... Denver, Colorado
O. Spurgeon English, M.D. ..... Philadelphia, Penn.
Temple Fay, M.D. ..... Philadelphia, Penn.
Hamilton Ford, M.D. ..... Galveston, Texas
Walter Freeman, M.D. ..... Washington, D. C.
Jas. K. Hall, M.D. ..... Richmond, Virginia
George T. Harding, M.D. ..... Columbus, Ohio
A. Hauser, M.D. ..... Houston, Texas
Eugen Kahn, M.D. ..... New Haven, Connecticut
Louis J. Karnosh, M.D. ..... Cleveland, Ohio
Jacob Kasanin, M.D. ..... San Francisco, California
H. Beckett Lang, M.D. ..... New York, N. Y.
Frank H. Luton, M.D. ..... Nashville, Tennessee
Wm. Malamud, M.D. ..... Worcester, Massachusetts
Donald McEachern, M.D. ..... Montreal, Canada
Karl A. Menninger, M.D. ..... Topeka, Kansas
Adolf Meyer, M.D. ..... Baltimore, Maryland
Glenn Myers, M.D. ..... Los Angeles California
Hans H. Reese, M.D. ..... Madison, Wisconsin
Paul L. Schroeder, M.D. ..... Chicago, Illinois
Theodore A. Watters, M.D. ..... New Orleans, La.
Wm. Walter Young, M.D. ..... Atlanta, Georgia

Titus H. Harris, M.D.
Galveston, Texas
EDITOR-IN-CHIEF

YEARLY SUBSCRIPTION $2.50
SINGLE COPIES 25 Cents

Murray Kornfeld
Chicago, Illinois
MANAGING EDITOR

WOODCROFT HOSPITAL

Pueblo, Colorado

Telephone 84

● *An institution for the diagnosis, care and treatment of those nervous and mentally ill and addictions.*

CRUM EPLER, Superintendent

COMPTON SANITARIUM
AND
LAS CAMPANAS HOSPITAL

COMPTON, CALIFORNIA

(Near Los Angeles)

Compton Sanitarium is a private psychiatric hospital with 120 beds. Emphasis is given psychotherapy by a staff of resident psychiatrists. Other treatment procedures are duly evaluated and administered. Careful records are maintained. There are psychiatrically trained graduate nurses; student nurses through affiliation; nursing attendants; dietitian; occupational therapist; recreational therapist; hydrotherapist; masseuse. Las Campanas Hospital (30 beds) provides adjunct general hospital facilities. Compton Sanitarium and Las Campanas Hospital are approved by the American College of Surgeons.

GLENN MYERS, M.D.,
Medical Director.

Beverly Hills Sanitarium

for

The Study and Treatment of Nervous and Mental Diseases

218 No. Westmoreland DALLAS, TEXAS Telephone 9-3114

This sanitarium is a psychopathic hospital with a home-like environment and is equipped to treat psychiatric and neurological cases in a most scientific manner by means of phychotherapy, occupational therapy, recreational therapy and physiotherapy, including hyperpyrexia, insulin and metrazol treatments in special conditions.

ARTHUR J. SCHWENKENBERG, M.D.
Attending Neuropsychiatrist

ROBERT E. WINN, M.D.
Resident Physician

VOL. II JULY 1941 NO. 7

EDITORIAL OFFICES
UNIVERSITY OF TEXAS SCHOOL OF MEDICINE
800 AVENUE B., GALVESTON, TEXAS

BUSINESS OFFICES
PHYSICIANS POSTGRADUATE PRESS
500 NORTH DEARBORN ST., CHICAGO, ILLINOIS

PRINTING OFFICES
ALAMOGORDO ROAD, EL PASO, TEXAS

DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

EDITORIAL COMMENT

The Battle of the Experts

Everyone is familiar with the American courtroom scene sometimes called the battle of the experts. Two doctors of equal prominence and ability are found pitted against one another in disagreement on a medical opinion. Is it any wonder that the general public is amazed and suspects that one or both are corrupt? The dignity of the medical profession has been offended. Its members have been embarrassed, most of them vowing that they will have nothing to do with the courts. The result—courtroom medical work falls into ill repute and is engaged in by physicians who are unscrupulous, or by members of other, less qualified, professions. This is true, not only in sanity hearings, but also in other court work.

Although this situation is lamented by everyone, very little is done to correct it. For although professions and concepts change, laws, like death and taxes, remain inviolate. And this procedure is based on the old English common law, conceived before medicine became a science. This law assumes that the practice of medicine is still but an art. It expects only an opinion from the physician and believes that this opinion can be formed from the evidence presented. It ignores the fact that medicine is a science, that it must not be limited by preconceived decisions of attorneys; that the doctor must be permitted to consider all the facts of the case and not only those which will further the wishes of the side by which he is employed.

As a scientist, the physician is not concerned with the outcome of the trial. He is not partisan in his interests. He is objective in his approach and scientific in his method of study. The law should permit him to use this scientific method and to be as impartial as the judge and the court.

In a few of our states, a fair and scientific basis for examination has been established. In New York state, the medical examiner system meets this problem. In Massachusetts, the Briggs law provides for criminal cases. An expert, who is legally known as amicus curiae, may be assigned as a friend of the court. However, this provision is rarely made use of in criminal proceedings where the question of sanity is raised. Another device used in some communities to meet this problem, may be likened to a court inquiry. Upon agreement of both attorneys, an impartial expert is selected whose report is presented to both of them and then introduced as evidence. As a general rule, however, only when the expert is paid by the government, are the results satisfactory. This method has been effectively used in Illinois for some time.

Eminent physicians have sometimes rejected invitations to study persons accused of crime because an impartial investigation is not possible. An outstanding example of this occured some years ago when a prominent psychiatrist declined an invitation from the defense to conduct a mental examination of two young men charged with murder. In this

(Continued to page 228)

The Recognition of Maladjusted Personalities by Draft Boards

HAMILTON FORD, M.D.
Galveston, Texas

Dr. Ford was graduated from the School of Medicine of the University of Texas in 1931. He received his post-graduate training in Neurology and Psychiatry at the Galveston State Psychopathic Hospital, the Department of Neuropsychiatry of the University of Texas, and the Section on Neurology at the Mayo Clinic. Dr. Ford has been Assistant Professor and later Associate Professor in the Department of Neuropsychiatry at the University of Texas and is associated in private practice with Dr. Titus H. Harris. He is on the Staff of the John Sealy Hospital and is Associate Consultant at the U. S. Marine Hospital. Dr. Ford was certified in Neurology and Psychiatry in 1939 and is a member of the American Psychiatric Association.

The importance of recognizing maladjusted individuals before induction into the armed forces is becoming increasingly evident. There are many persons who are so poorly endowed or possess such a poor personality make-up that they have been unable to adjust to the requirements of life and have reacted in such a manner as to become a problem to friends and acquaintances. In particular, reference is made to the severe mental defectives, the frank psychoses, the chronic psychoneurotic invalids, and those neurological disorders which are accompanied by mental symptoms.

The term "maladjusted" in the title may be misleading and cause one to think only of this group of illnesses. While this personality should be included in order to eliminate them before induction, we are more interested in finding ways to recognize that person whose behavior is on the borderline between normal and abnormal adjustment, or who will become a problem in the Army when faced with many difficulties.

Draft boards were concerned only with the physical fitness of the candidate during the earlier months of World War I. This occurred in spite of the experiences of our former allies, for strange ailments had begun to occur in their armed forces shortly after the start of the war.[1] At first these conditions seemed to appear only in soldiers who had been in battle, and it was felt that some structural change in the brain was responsible. The term "shell shock" was coined from this theory. Soon similar disorders were observed in soldiers who had never been near an explosion; thus the original postulate had to be discarded. Intensive studies next revealed that personality inadequacies existed in each one and necessity called for a more complete understanding of specific cases. This was the stimulus that caused psychiatry to attain the position of prominence which it occupies today.

Even though one-seventh[2] of all discharges from the British Army during that war were due to mental and nervous diseases, psychiatrists on the advisory board and in the armed forces did not receive full cooperation of army officers because many men whom they had rejected were continued in the services. Quite pertinent was a cablegram from General Pershing to the War Department in July, 1918,[1] which changed the status of things for he stated, "Problems of mental disorders in replacement troops recently received suggest urgent importance of intensive effort in eliminating mentally unfit from organizations (in the) new draft prior to departure from the United States."

The Medical Department of the United States Army published in 1929 an interesting survey of neuropsychiatry in the World War[1] which sheds much light on the problem of mental and nervous illnesses, first to show the prevalence of these disorders in the men actually in military service and secondly to illustrate the frequency of occurrence of the various types that are going to require special attention. These figures apply only to an approximate 3,500,000 men in the home forces who received neuropsychiatric examinations after they had entered the military service, and *do not* include the large group of men who were deferred by draft or advisory boards once they had begun to function properly.

Table I shows that 69,394[1] soldiers were found to have mental and nervous disorders which constitutes a ratio of 20 per 1000 soldiers.

When grouped by diagnosis the psychoses with 11.4 per cent, nervous diseases and in-

juries with 10.0 per cent, epilepsy with 9.2 per cent, and the endocrinopathies with 6.9 per cent, totalling 37.5 per cent, make up one-third of the illnesses, and the local medical examiner for the draft board should defer them into Class IV just as instructed by paragraph 77 of *Selective Service Regulations,* Vol. VI entitled *Physical Standards.*[3] Recognition of this person is all that is necessary since the medical examiner must defer this maladjusted personality or refer him to the psychiatrist on the Medical Advisory Board. He is not required to make a diagnosis of dementia praecox or manic-depressive psychosis; consequently a broad knowledge of psychiatry is not required. As a rule their behavior and expression of delusions or hallucinations will identify them, but if not, the methods of study to be outlined later will suffice to pick out these draftees.

Thirty-one and five-tenths per cent were feeble-minded, and this prevalence of mental deficiency was responsible for the early utilization of intelligence testing by the army. The majority of the feeble-minded group were of the moron level, which means that they were near the borderline of normal and were passed by the medical examiner at induction because no special effort was made to identify them. If the borderline feeble-minded are added to the other maladjusted personalities likely to escape recognition (see list in lower right hand corner of Table I), i.e., the psychoneuroses comprising 16.5 per cent, the constitutional psychopathic states 8.9 per cent, drug addiction 2.9 per cent, alcoholism 2.7 per cent, this reaches a total of 62.5 per cent which require a rather searching examination to identify them. Incidentally, many of the cases then classified under the endocrine and neurological disorders, such as neurocirculatory asthenia and the various tics, are now recognized as psychoneuroses, and might further add to this total group of mildly maladjusted men who had not only passed the medical examiner on the draft board, but also the examination when inducted into the army.

This 62.5 per cent constitutes the greatest problem in recognition for the draft boards because they usually have made fair citizens and can continue to function normally or work gainfully in some industry as a civilian. There is an inability, though, to adjust to the difficulties confronting them in the army,[4]

TABLE I

NEUROPSYCHIATRIC CASES IN 3,500,000 SOLDIERS

Diagnoses	*No. Cases*	*Per Cent*	5	10	15	20	25	30
Mental Deficiency	21,858	31.5	******	******	******	******	******	******	**
Psychoneuroses	11,443	16.5	******	******	******	**		
Psychosis	7,910	11.4	******	******	*			
Nervous Diseases and Injuries	6,916	10.0	******	******				
Epilepsy	6,388	9.2	******	*****				
Constitutional Psychopathic States	6,196	8.9	******	****				
Endocrinopathies	4,805	6.9	******	*				
Drug Addiction	2,020	2.9	***					
Alcoholism	1,858	2.7	**					
TOTAL CASES	69,394	100.0						

Mental Deficiency	31.5%
Psychoneurosis	16.5%
C. P. I.	8.9%
Addiction	2.9%
Alcoholism	2.7%
TOTAL	62.5%

RATIO OF 20 CASES PER 1,000 INDUCTED

Modified from Chart I, Diagnoses of Neuropsychiatric Cases, The Medical Department of the U. S. Army in the World War, Neuropsychiatry, Vol. X:157, 1929. Government Printing Office, Washington, D. C.

such as separation from family ties, submission to discipline, a change in daily routine with a new occupation, forced restrictions, barrack life, and the requirements expected of them as a unit in their company. Another big precipitating factor is the threat to the soldiers' primary instinct of self-preservation by constant danger. Such people, when left to their own devices in civilian life,[5] have been able to hide their eccentricities and conflicts through compensatory means arising from the freedom which allows them to choose their way of living.

Shortly after the war these seventy thousand ex-service men made up 22 per cent of all disabled veterans and by 1927, this proportion had increased to 46.7 per cent,[1] and more recently to more than one-half[5] of all patients receiving hospital treatment from the United States Veterans' Bureau. If these facts were transformed into loss of efficiency or considered in terms of waste,[1] we must think not only of the tremendous financial outlay for hospital care and compensation since the war, or the cost of care during the war and the cost of equipment and maintenance, but also of the waste of time of their instructors, the interference in training of other soldiers, and the general breaking down of morale in the troops associated with them.

A comparison of neuropsychiatric illnesses with others in soldiers shows why the psychiatrist was considered to be secondary only to the eye, nose, and throat man on medical advisory boards.[6] The War Department is concerned chiefly with the establishment of an efficient fighting force and to do so capable soldiers are necessary. The survey just quoted has shown that 96.1 per cent[1] of the cases of nervous and mental diseases were not due to disability resulting from service in the army; rather the cause existed prior to induction. The soldiers in the Canadian Army service forces of World War II had only superficial psychiatric studies and Baille[7] in a recent report based upon a study of 200 invalided neurological and psychiatric hospital admissions estimated that 34 per cent should have been recognized at induction.

Not to be caught napping the War Department prior to the passage of the Selective Service Act asked the assistance of psychiatric organizations to find methods to prevent these mistakes, as well as those of the last war. Statistically, nineteen of every twenty neuropsychiatric invalids could be eliminated before induction; actually, this is too much to expect. There are not enough qualified psychiatrists in the United States to have one on each of the 700[8,9] Medical Advisory Boards. Thus the medical examiners of the 6,500 local boards must function as psychiatrists and be very much on the alert. The purpose in presenting statistical data is to make the physician who serves on the draft board aware of the immensity of the problem, for only in this way will interest be aroused and the proper attention given to the recognition of maladjusted personalities. Exclusive of the frank nervous and mental diseases, one authority[5] has stated that 5 per cent of all draftees should be referred to the consulting psychiatrist on the medical advisory board; otherwise the examinations are inadequate. The government has promised a psychiatrist at each induction center to examine fifty draftees a day[8,9] and while it is possible that the person who is psychically unfit will be found, the rejected man is burdened for he has given up his job, and the expense to and from the induction center is no small item.

The recent psychiatric literature contains very little that will show how to recognize this particular group. The medical circulars distributed by the National Headquarters for the Selective Service System[3,10] offer very few suggestions of a practical nature that will help us identify the inadequate, poorly adjusted person. It would be relatively easy to prepare an examination to be utilized by trained psychiatrists who could devote from 15 to 30 minutes to neuropsychiatric case study, but it is very hard to prepare such an outline for a physician who has but a few minutes for the study and is unaccustomed to thinking in terms of personality factors. Fortunately there are a few things which can be done to make this task easier. In the first place, the local draft board has one advantage since the draftee usually comes from their district and the board either has some knowledge of the individual or can interview acquaintances of the candidate if suspicions are aroused about his fitness as a soldier. This is important and should be utilized to its fullest extent. Next, the task might be simplified if the State Director of Selective Service would compile a list of all men for that par-

ticular state or region in the draft age who have been in either state or private mental hospitals, and furnish this information to each local board. This should not entail very much effort. Another factor which should make the task simpler than in the last war is the well organized Selective Service set-up originally planned for peace time conscription that caused every man of draft age to be registered by his local board, and this will prevent draftees who have been rejected from going to other places for examinations. A proposal of educational value is the plan to hold regional seminars in psychiatry,[8,9] which will be available, not only to psychiatrists on the medical advisory board, but also to medical examiners who can be given some instructions in the fundamental principles of understanding personality structure.

Identification of the maladjusted personality may not be so difficult if we remember that the prime purpose of any examination is to gain a complete knowledge of that person, which includes not only a general examination of his physical being, but also information pertaining to his past health and adjustment and how he has behaved as an individual in his social surroundings. An understanding of the type of personality can be gained in the majority of cases by simple history of his past performance in life and a few observations made during the course of the routine examination, which can very easily be made a part of the regular examination form issued by the War Department.

Tables II, III, and IV will serve as a brief outline for an examination that will disclose the majority of the poorly integrated men why may be expected to react to the stress of army routine and the threat of war with a mental illness. For convenience the outline can be placed on a sheet of paper for reference until the examiner has become familiar with the routine through frequent repetition. Naturally it is not necessary to delve into each phase with every patient, but a general broad inquiry, coupled with your observations, should suggest the presence of some abnormality; then it will be advisable to question the candidate at greater length. Numerous investigators have shown that a few questions pertaining to the school history and occupation would give enough information to eliminate a large number of men. For instance, the studies of Baille[7] on the invalided Canadian troops revealed that 132 of the 200 soldiers did not finish high school and 51 per cent had a bad industrial record.

TABLE II

A BRIEF METHOD OF PERSONALITY STUDY

A. *History Suggestive of Poor Endowment.*

1. Education
2. Occupation
3. Family
4. Delinquency
5. Illnesses
6. Development
7. Social
8. Sexual
9. Habits and Ideals

B. *Behavior Suggestive of Instability.*

1. General Attitude
2. Behavior
3. Speech
4. Understanding

Table II shows the general heading for the psychiatric study. A brief method of personality study requires certain facts in addition to the school and work history, which include the family set-up, the delinquencies, past illnesses, development, social and sexual adjustment, and the habits and ideals of the proposed soldier. During the examination types of behavior that show instability are noted under general attitude, speech, behavior, and the degree of understanding shown about himself.

The following tables will show these individual facts in greater detail. A glance at Table III reveals questions pertaining only to things in the history[11,12,15,17] of the draftee that would show a poorly integrated personality; conversely their absence means a more stable person. If the history alone is considered, a draftee with several inadequacies scattered throughout the personality study would point to a maladjusted personality. This has been illustrated by underlining some positive historical facts showing a probable constitutional psychopathic inferior; however, as previously stated, there is no need to identify these milder personality disorders in specific reaction types. Nevertheless, the findings automatically fall into categories and will be considered in this way for convenience.

The constitutional psychopathic inferior is usually very intelligent, and may be a college graduate, but there are frequent job changes, few promotions or salary increases, and more important, seldom a bank account. Also he may be the type who has received compensation insurance or has come from a broken home, with some maladjustment in parents

TABLE III

A BRIEF METHOD OF PERSONALITY STUDY

History

EDUCATION	OCCUPATION
Grade attained	Type work
Age on leaving	*Duration*
(why)	Advancement
Failures	*Salary, savings*
	Compensation
FAMILY	DELINQUENCY
Mental Illness	*Truancy*
Crime	Vagrancy
Economic Status	Arrest
Broken home	*Misdemeanors*
Only child	
Spoiled child	
PAST ILLNESSES and	COMPLAINTS
Injury, delirium	Seen nerve doctor
Fits, encephalitis	Seen chiropractor
Institutionalization	Concern with bowels
Frequent operations	Concern with food
Long convalescences	Concern with self
Frequent complaints and	told nothing wrong
DEVELOPMENT	SOCIAL ADJUSTMENT
Slow to walk and talk	Poor mixer
Tantrums, sleep	Dislikes people
Bedwetting	*Not liked by people*
Nightmares	Unfairly treated
Stammering, nail biting	"Butt" of jokes
SEXUAL ADJUSTMENT	HABITS AND IDEALS
Deny masturbation	Religious Beliefs
Experiences	*"Against" customs*
Number "dates"	*Drugs, alcohol*
Marriage	No. of habits (bad)
Divorce	*Inability to change*

and siblings, and most often is a spoiled child. Truancy is a frequent finding, and a history of misdemeanors is common. Temper tantrums probably occurred, and bed wetting lasted past infancy. Friends are easily made but not retained, and unusual sexual experiences, even perversion, may exist. Divorce can be a factor here. The man is usually opposed to everything and shows very little ability to change his habits or ideals. A history of narcotic addiction, the use of sedatives, or over-indulgence of alcohol is highly significant.

Table IV on behavior shows that he probably has a rather good general attitude and appearance, and makes in fact a favorable but deceiving first impression. Aggressiveness, braggadocio, or resentfulness are frequent traits coupled with a tendency to talk a great deal about himself. He will not realize his shortcomings or his mistakes, and his patriotism can be questioned because he has always thought only of himself.

The mental defective on the other hand can be identified in 80 per cent of the cases by asking about the grade attained, the age of leaving school, ease of learning, longest period of job holding, promotions received, tramp-life or arrests, frequent injuries or bed wetting. The answers will readily stamp the level of former adjustment. On observing him, the examiner would note that he was careless in his dress, silly, moved slowly, appeared perplexed, talked in a hesitant manner and cooperated very poorly for the examination.

Because of the youthfulness of the draftees, organic brain disease leading to personality changes could be detected by asking about spasms or convulsions, delirious states, sleeping sickness, head injuries, or stigmata in the antecedents.

The man likely to develop a psychoneurosis usually has a good school and work history, but can have a family history of mental or nervous illnesses or a broken home. This history includes frequent minor operations, prolonged convalescences, and many complaints. Concern is expressed about the food he eats, his physical status and bowel function. If a "nerve doctor" or chiropractor have not been consulted, certainly some physician has told him that "nothing is wrong with you" during one of his many clinical investigations. On examination this person will appear tense, tremulous, sweating, will have "bitten fingernails," will be ill at ease, but will have a realization of his shortcomings.

The rather rigid personality has been a very conscientious worker, has never been in trouble, is concerned perhaps with his phys-

TABLE IV

A BRIEF METHOD OF PERSONALITY STUDY

Behavior

1. General Attitude	*2. Behavior*
Careless or unusual dress	*Aggressive*
Silliness	*Boastful*
Meaningless actions	Bubbling over
Tremor or sweating	Perplexed
Shyness on exposure	Ill at ease
Slow movements	Suspicious
Rapid movements	*Resentful*
Disfigurement	Despondent
3. Speech	*4. Understanding*
Constant talk	Stupidity
Rambling	Forgetfulness
Unintelligible	Illiterate
Hesitant speech	*Realizes shortcomings?*
Talk of self	*Realizes mistakes?*
Abnormal trends	*Patriotism?*
Impediment	

ical functions, and may have been a bed wetter or stammerer. He is a poor mixer, has never had sexual contacts, has rigid ideals and beliefs, is totally opposed to alcohol, and experiences pronounced fluctuations of mood, but will probably show nothing much on examination except shyness when exposed in the nude around many other men.

These are only a few examples illustrated by such an outline for personality study and the types of personalities to be encountered by medical examiners of draft boards. Thus is a basis found to identify the men who may have difficulty after induction into the army.

Certain authorities question the advisability of deferring this group, but the experiences of the last war taught that 96.1 per cent[1] of similar cases should be detected before induction and has caused the medical department of the army to feel that there are sufficient men with sound physical health and emotional stability to fulfill all the needs of the armed forces. The old idea of placing a bad boy in the army in order to make a man out of him is entirely erroneous, and one good authority has answered the question by stating that there are no halfway measures in the armed forces; either a man is fitted or he is unfitted for military service.[1] If unfitted, he will be an economic loss and a handicap, but if deferred he could be made to serve a useful purpose in defense industries.

One would feel that there are other reasons to exclude men with poor temperaments. In the first place, the newer mechanized type of warfare will demand the utmost in precision and ability from the individual since he must be able to coordinate with the company as a whole for smooth function. Also the men who have been conscripted into the army entered with the idea of spending a year in training for defense of the country; consequently their attitude will be different from that of the soldier in World War I, not only toward a possible war, but to life in general.[13] They are the products of the economic depression and have seen old ideals of decades disproved. Their opportunities were rare, restlessness prevails, and they are eager for a change, are less suggestible, and may not be easily aroused to heights of patriotism through methods used in past wars. As a consequence a more fertile field exists for the development of mental disorders.

When this discussion was first considered there was a plan to induct only 1,000,000 men for training. The plan now is to draft 3,000,000 more men, and if the figures previously given on the last war are adhered to, i.e., a ratio of 20 men per 1,000, the army can expect 80,000 men with neuropsychiatric illnesses of such severity to cause discharge from the army unless they are recognized and eliminated early. Also, there has been a gradual, but nonetheless purposeful, step - by - step march toward participation in another war. This first began with an alteration of the Neutrality Act and has now reached the point of furnishing other than economic aid to the forces opposing the axis. If war comes, not only the soldiers, but the civilian population,[13,16] will be subjected to more stress because of the type of warfare, with the probable occurrence of more neuroses in both groups unless our present method of rationalizing the position of the United States in world affairs will suffice to create the necessary morale which will enable the soldier and the civilian to forget the threat to their lives. There must be no skepticism about the aims and purposes of this war, for the unstable soldier, faced with all of this, will have more possibilities of reacting to these changing factors by the development of personality illnesses.[14] Under such circumstances, immediate measures should be taken by the medical examiners of draft boards to recognize the maladjusted personalities before passing the men for induction. Unnecessary waste, suffering, and expense could thus be eliminated.

References

1 The Medical Department of the United States Army in the World War, *Neuropsychiatry*, V. 10: 2, 1929. *United States Government Printing Office*, Washington.
Ibid V. 10: 57, 1929.
Ibid V. 10: 157, 1929.

2 House, William, *Northwest Med.*, V. 17: 262, 1918.

3 Physical Standards, Selective Service Regulations, V. 6, 1940.

4 Cohen, S. A.: *War Medicine*, V. 1: 205, 1941.

5 Bowman, K. M.: Ibid, V. 1: 213, 1941.

6 Harris, M. L.: *J. A. M. A.*, V. 70: 157, 1918.

7 Baille, William: *Am. J. Psychiat.*, V. 97: 753, 1941.

8 Southern Psychiatric Association Report of its Committee on Psychiatry and the National Defense, *Psychiatry*, V. 3: 619, 1940.

9 Psychiatric Mobilization in the United States

Army, *Am. J. Psychiat.*, V. 97, Supplement.
10 Selective Service System, National Headquarters, Washington, D. C., 1940.
11 Stearns, A. W.: *Mil. Surgeon*, V. 43: 652, 1918.
12 Report of the Military Mobilization Committee, *Am. J. Psychiat.*, V. 97: 475, 1940.
13 Sullivan, H. S.: *Men. Hyg.*, V. 25: 5, 1941.
14 Benton, G. H.: *J. A. M. A.*, V. 77: 360, 1921.
15 Stearns, A. W.: *J. A. M. A.*, V. 70: 229, 1918.
16 Sands, I. J.: *War Medicine*, V. 1: 203, 1941.
17 The William Alanson White Psychiatric Foundation Bulletin: "A Minimum Psychiatric Inspection of Registrants," *Psychiatry*, V. 3: 625, 1940.

The Psychologic and Economic Influence of Alcohol

H. MASON SMITH, M.D.
Tampa, Florida

Dr. Smith received his medical degree from the University of Georgia Medical School in 1908. He is Head of the Section on Neuropsychiatry at St. Joseph's Hospital and Tampa Municipal Hospital and is Attending Specialist at the Veterans Administration Facility. Dr. Smith is a Diplomate of the American Board of Psychiatry and Neurology, a Fellow of the American College of Physicians, and a member of the Southern Psychiatric Association.

During recent years, interest and attention have been increasingly directed toward alcohol. In the medical literature reports by research workers as well as clinicians are abundant. The problem of confirmed and chronic alcoholic intoxication is apparently growing.

Practically all civilizations have been influenced by the effects of alcohol. In his article entitled *Alcoholism and Its Effect Upon Body Systems,* Loeb[1] cited Samuelson's observation in his *History of Drink,* that in every civilized nation the time of highest culture is preceded and then followed by a period of chronic alcoholism. About 500 B. C., during the height of China's culture, Mensius, a disciple of Confucius, expressed the fear that his country was reverting to the drunkenness and gross intemperance prevalent in the period before his time. History attests that these conditions continued concurrently with the decline that followed. In India, as in China and other Eastern countries, a like trend associated with the widespread use of alcohol culminated in stagnation and decadence. Greece offers a classic example among Western nations of bacchanalian revelry interrupted by the relative temperance of the period of highest culture; but with alcoholism and dissipation again in the ascendancy her declining civilization was eventually merged into the Roman. Instances depicting the same wavelike trend dot the pages of history, apparently even contemporaneous history.

A United Press report of August 24, 1940, from Vichy, France, stated that the Government attributed the moral collapse of France under the German attack chiefly to alcoholism, the worst of four problems, the lesser ones being tuberculosis, cancer and syphilis. This same dispatch disclosed that drunkenness was rampant in the Army during the eight months of inactivity early in the war; that in a single hospital in the area of the Fourth Army 814 cases of delirium tremens had been treated during the month of January; and that the disastrous era of intoxication had caused most of the nervous breakdowns and shell shock among young French soldiers when they had had to face dive bombers and tanks. It appears from this information and other facts that the alcoholism and sensuality attendant upon revelry at the shrine of Bacchus have been coincidental with the decadence of the French nation today.

The pathologic changes resulting from acute alcoholic intoxication and poisoning are now better understood. With the aid of spinal drainages and hypertonic solutions the edematous brain is reduced. With the advent of synthetic vitamin B_1 and insulin, the system is quickly detoxicated and the metabolism improved, and with the use of benzedrine sulphate the post-alcoholic emotional slump is detoured. In fact, the science of medicine has so streamlined the treatment that it is now possible for the industrious business man to recover from his week-end spree on Sunday night, return to his office on Monday morning and work with energetic speed so that he may begin early the following week-end on the next spree. However, at a period in ratio to the stability of his

nervous system, he loses his efficiency and becomes a problem drinker. He indulges in sprees of longer duration; he experiences more difficulty in becoming sober; and after each subsequent remission he shows less dependability and efficiency, thus bringing upon himself and his family more and more domestic problems.

The psychiatrist finds that such a patient is suffering at this stage from a subclinical or minor psychosis, or is afflicted by some form of neurosis, growing out of a bad family or childhood background. The episodic recurrence of a manic urge with loss of inhibition, or a depression with failure to find interest or pleasure, causes this type of drinker to seek euphoria and happiness, or complete escape into oblivion.

It is assumed that some men of the extrovert type drink to escape situations, such as may be brought about by a business worry, a nagging wife, sleeplessness and various other difficulties. It is undoubtedly true that many of the problem drinkers have been overly protected and overindulged in childhood, growing up to be selfish and spoiled, while domineering fathers and indulgent mothers waged many a conflict over their management. Others of the asthenic or somewhat shut-in type, disturbed by somatic sensations of emotional origin, have a definite sense of inferiority and take liquor in solitude to overcome this feeling of inadequacy.

Tucker[2] concluded that among drinkers there is a group of both periodic and chronic alcoholics who suffer from disturbed metabolism in the form of a spontaneously occurring hypoglycemia. In these patients, he observed, a sufficient amount of carbohydrates not only meets the deficiency of blood sugar but also controls their alcoholic propensities.

Stevenson[3] regarded addiction to alcohol as "a special type of psychoneurosis, based on a special type of personality, difficulties of adjustment, habituation, and just as definitely motivated, the patient usually not being aware of his motives, but tending to offer a variety of rationalizations for his behaviour." In dealing with the problem drinker, he found it essential during treatment hours to endeavor "to get a full understanding of all the personality factors that are involved, to explain to the patient the real motives behind his rationalizations, and to re-educate him through progressively higher levels of adjustment, aided by the learning of new and better habits, new and better escape mechanisms, to a level where he has a good understanding of all these factors and at which alcohol no longer has a real appeal to him. That this is not an easy task goes without saying."

Success in treating a number of problem patients was attained by McConnell[4] through psychotherapy, continued in a series of interviews at the office after the patient had been discharged from the hospital. First endeavoring to ferret out the patient's intolerable situation, he then set about regulating his occupational and physical activities while he further studied his mental reactions and gave him moral support.

Although many cures of alcoholism have been effected through religious influences and some through suggestion made by charlatans and quacks, such as the old Keeley cures, the scientific approach to the problem consists in obtaining an understanding of the personality make-up, correcting the maladjustment and eliminating the mental conflicts. The books of Emerson[5] and Strecker[6] have presented the method by which these ends may be most skillfully accomplished. Strecker[6] recommended psychotherapeutic conversations covering 100 one-hour periods during a year, at the rate of three a week with longer intervals intervening as the patient improves. Because of the increasing number of problem drinkers, this method of therapy is physically impossible of application to the large numbers who need it. To only a few who are well-to-do is it available. A very small number of our state institutions are staffed so as to do even a minor part of this needed time-consuming therapy and, too, when the drinker reaches an institution, his nervous system has usually undergone organic changes to such an extent that this type of treatment would be futile. How many psychiatrists would the French Army have needed when there were 814 cases of delirium tremens in only one hospital during one month? Thus it becomes apparent that the problem has to be considered from other angles also.

Aside from neuroses and psychoses, a factor in alcoholism well worth consideration was presented recently by Myerson.[7] In discussing the social psychology of alcoholism, he recorded interesting observations on Jews

and alcoholism and on women and alcoholism. Surely every reader has noted the frequency of neuroses among Jews and also the abundance of somatic sensations experienced by them. They have their full share of feelings of inferiority and depression and are by no means immune to psychotic disorders. Psychologically and physiologically they respond to alcohol as do other races. Nevertheless, they do not drink to excess, and alcoholic mental disease is rare among them. Myerson[8] cited Pollock's report of 546 first admissions of alcoholic patients, only four of whom were Jews, and further cited Krinsky's statement that of 860 patients with delirium tremens admitted to the Boston Psychopathic Hospital between 1913 and 1938, not one was a Jew. Observing that Jews are rarely total abstainers and have neither total abstinence societies nor even temperance societies among them, he concluded that the explanation of this phenomenon lies in the social tradition of the Jew in respect to alcohol. "To be a drunkard," he related, "is to cease being a Jew, is to leave the fold of Jewry or to be pushed out," for "nowheres and amongst no people is there more contempt, loathing, and even hatred for the alcoholic."

Turning to women, who constitute one half of mankind, this author noted that the total incidence of alcoholism is much lower among them than among men, available statistics indicating that the proportion is about 1.7 per cent; nevertheless, the incidence of neuroses, feelings of inferiority, difficulties of adjustment, particularly sex maladjustment, manic-depressive psychoses and dementia praecox, and the various non-organic mental diseases is fully as great, if not greater, among women. He observed further that only in the realm of the alcoholic and syphilitic psychoses do men excel them in getting into mental trouble. Again, as with the Jew, he found in "a social tradition of enormous validity and importance" the factor preventing or minimizing alcoholism among women, for society punishes the female even more than the male, and the woman who got drunk, until recently at least, was ostracized as an abandoned creature.

The question of why people drink excessively has been discussed at length. Certainly the Jewish people set an example that may well be emulated by the nations of the world, particularly France, whose excessive consumption of alcohol per capita is indicated in Table I. It is a matter of regret that available statistics are meager and fragmentary at best, constituting only approximately accurate estimates of the per capita consumption of this drug.

The French have long been noted for their pursuit of bodily pleasures. They are known as the world's greatest connoisseurs of food and, also, according to general impression, as the greatest gourmands. Judging from the literature of the French writers, they have been the most sensual.

French styles, French cuisine, French Follies and the fruitage of bacchanalian pursuits have long dominated the French scene, at least in the eyes of a world that has through the years looked upon Paris as the paradise of sensual delights. With few exceptions, the French have played a very inconspicuous part in sports and athletics. Their representation at the Olympic games has been surprisingly small, and their activities in athletics have been inconsiderable as compared with other nations.

In 1938, to the casual observer it appeared that the greater part of the population spent its time in the sidewalk cafes drinking wine and indulging lavishly in the feeling of security that the Maginot Line provided. The tourist with a small amount of baggage was inconvenienced because of the short hours for all workers, and laundry could be obtained only once a week. Then with a forty-hour week, while the Germans were working night and day at war preparations, and with mobilization of soldiers taking place at the moment, defense workers in Marseille struck for still shorter hours and more pay. To me it appeared that the same pleasure-craving impulse that caused the French people to avoid work and make a ritual of eating, also made them seek euphoria through drinking wine all day long. Sensuality and laziness are not classified as neuroses.

It is not my purpose in this paper to go into the pathologic changes caused by alcohol, but in this connection the effect of the drug on the hypothalamus should be mentioned. Gantt[9] demonstrated that alcohol has a depressant and toxic effect on the cerebral cortex despite a concurrent accelerant influence on the cerebral blood flow. Recently,

Masserman and Jacobson[10] reported that the intravenous administration of from 1 to 3 cc. per kilogram of absolute alcohol increased the reactivity of the hypothalamus in cats while depressing both the motor and the vegetative functions of the cortex. They also demonstrated the obverse of the normal relationship in that the hypothalamus influenced the cortical action. Their experiments showed that "localized faradic stimulation of the hypothalamus in the intoxicated animals not only induced the usual transient vegetative and motor emotional mimetic reactions, but also initiated the more complicated, maintained and directed defensive and offensive behavior in which, according to present concepts of the integration of central nervous activity, the cortex necessarily participates. In contrast to the motor coordinating functions of the cortex, however, its temporal inhibitory influence on lower centers was apparently impaired in the intoxicated animals, permitting the emotional mimetic behavior to continue for longer periods than in the normal controls."

Tucker[2] expressed brilliantly the hazards of behavior when emotions are unrestrained by reason, that is, when the cortical inhibitions on the basal ganglia of the brain are lost. He described the most effective individual as the one who under stress envelops the rising emotions with the reasoning faculties, holding them in abeyance in order to direct them with the rational purpose and good judgment that lead to sensible accomplishment, thus conditioning his emotions. Also, he pronounced emotions as contagious as measles and pointed out that uncontrolled mass emotion culminates in the unreasoning fury of the mob, leading to all kinds of violence, even war. He concluded that the imbalance between reason and the emotions, between the cortex and the hypothalamic areas, may be functional or organic and may have its origin in misdirected or perverted instincts, bad heredity, toxic or degenerative changes of the brain, innate or acquired hypersensitivity or hyposensitivity of the emotional system and other causes.

It is hardly to be disputed that the emotion of fear can permeate an army and bring into play the instinctive urges of escape and submission. Personal observations of the drinking habits of the French people, supported by the dispatches from Vichy and the meager data presented in tables, point to a loss of cortical influence on the hypothalamic areas of the brain in a vast number of the soldiers of the French Army, a condition which was productive of war shock and defeatism. The evacuation of Dunkerque, one of the greatest tactical feats in history, was in contrast to the French debacle an admirable example of conditioned emotions of men under greatest stress held in control by reason while judgment directed their activities and their fate.

In the United States the per capita consumption of alcohol increased rapidly each year from 1933 through 1937. In 1938, it was 14.33 gallons, about half a gallon less than in 1937, and in 1939 it declined somewhat; but it is still so great that it should now be the subject of earnest consideration. Repeal has surely demonstrated that prohibition was not the instrument whereby control could be made effective, but legislation of an appropriate type could make intelligent temperance laws really helpful. The flow of alcohol should be controlled and handled in such a way as to protect the youth. Laws backed by an educated public opinion and designed so as not to antagonize psychologically the consuming public could attain this objective.

Despite none too effective educational efforts of the past, education seems to me to hold the greatest promise as a means of combating the encroaching and increasingly destructive effect of alcohol on the individual and national life. Where the moral, sociologic and economic interests of the nation are so vitally concerned and so gravely affected, the people should be informed. Studies of the physiologic and psychologic effects of alcohol should be required of the students in every college and of the soldiers in every training camp so that they may appreciate the subtle, habituating and destructive influence of this drug on the personality. The press and federal agencies have successfully disseminated information as to the methods and technique of the German propaganda, in itself as subtle and insidious and as filled with false hopes and promises as the euphoria of alcoholism, also quite as destructive to the political integrity of the nation as alcohol is to the personality. The aid of these and all other available agencies might well be enlisted in the interest of the national welfare

to disseminate information as to the true nature of alcohol and its effect upon the human body.

1019 Citizens Building.

References

1 Loeb, William A.: "Alcoholism and Its Effect Upon Body Systems," *M. Bull. Vet. Admin., 16:* 258-267 (Jan.) 1940.
2 Tucker, Beverley R.: "Alcohol or Candy," *Virginia M. Monthly, 66:* 329-332 (June) 1939; "The Emotion-Reason Balance," ibid. *65:* 128-131 (March) 1938; "Emotional Illness: Pathways, Manifestations, and Treatment," *Diseases of the Nervous System, 1:* 168-174 (June) 1940.
3 Stevenson, G. H.: 'The Psychological and Medical Aspects of the Excessive Use of Alcohol," *Canad. M. A. J., 42:* 57-61 (Jan.) 1940.
4 McConnell, Whitman Carlisle: "Treating the Problem Drinker," *M. World, 58:* 301-303 (May) 1940.
5 Emerson, Haven: *Alcohol and Man,* New York, Mcmillan Company, 1932.
6 Strecker, Edward A. and Chambers, Francis T., Jr.: *Alcohol One Man's Meat,* New York, Mcmillan Company, 1938.
7 Myerson, Abraham: "The Social Psychology of Alcoholism," *Diseases of the Nervous System, 1:* 43-50 (Feb.) 1940.
8 Sources: Emerson's *Alcohol and Man,* p. 426; articles by Dr. R. C. Hercod, Secretary-Director of the International Temperance Bureau, Lausanne, published in *The Voice* of the Board of Temperance of the Methodist Church, and other material published therein.
9 Gantt, W. H.: "Effect of Alcohol on Cortical and Subcortical Activity Measured by the Conditioned Reflex Method," *Bull. Johns Hopkins Hosp.,* 56: 61 (Feb.) 1935.
10 Masserman, Jules H. and Jacobson, Leon: 'Effects of Ethyl Alcohol on the Cerebral Cortex and the Hypothalamus of the Cat," *Arch. Neurol. & Psychiat., 43:* 334-340 (Feb.)

TABLE I—CONSUMPTION OF ALCOHOLIC BEVERAGES[8]

Per Capita in Liters

	Beer		*Wine*		*Spirits*		*Absolute Alcohol*		
	1928-32	*1938*	*1928-32*	*1938*	*1928-32*	*1938*	*1920-22*	*1928-32*	*1938*
France	33.70	27.89	41.75	137.55	5.22	2.84	17.61	20.64	17.55
Gr. Britain		43.13		1.37	1.29	0.83	6.16		3.51
Germany	76.77	63.70		5.00			2.66		1.21
Italy	1.91		94.00		0.09		13.77	11.18	
Norway	17.04		1.66		2.12		1.99	2.11	
Sweden	25.20*		0.90*		4.40		3.03		

**Average for 1926-1930.*

Résumé of Metrazol-Shock Therapy in Schizophrenia

SERGE ANDROP, M.D. *and* ETHEL GREEN, M.D.
Catonsville, Maryland

Dr. Androp was graduated from Loyola University School of Medicine in 1913. He also possesses the degrees of L.L.B., and D.C.L. He is a diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association. He is Assistant in Neuropathology at the Johns Hopkins University and a member of the staff of Spring Grove State Hospital. He is the author of several articles on psychiatry and neurology and was the recipient of the first prize and $3,000 for his work on the genetics of mental disorders from the Eugenics Research Association in 1935.

Dr. Green was graduated from the University of Buffalo School of Medicine in 1920. She is a member of the staff of Spring Grove State Hospital.

In 1935 Meduna[1] introduced the treatment of schizophrenic patients by the provocation of convulsive seizures with metrazol on the assumption that there is a biological antagonism between schizophrenic processes and epilepsy. This hypothesis was established on two factors: First, anthropometric investigations of Kreyenbruck, Graf, Delbruck, and others pointing to the fact that the diseases are usually associated with different constitutional bodily types—the athletic and displastic types predominating in epilepsy, the asthenic-leptosome constitution in schizophrenia. The second and more fundamental factor is found in the works of Muller, Glaus, Steiner, and Straus, emphasizing the rarity of the combination of schizophrenia with epilepsy.[26] It is the writer's opinion that data are not sufficient to make a valid conclusion concerning the possible antagonism between the two diseases since the hypothesis has

not yet been proved.

Kraepelin[2] refers to epileptiform attacks or spells of dizziness and fainting in dementia praecox, giving the frequency of such cases as 16 per cent in his Munich clinic and 19 per cent in the Heidelberg material.

Vorkastner believes in the concomitant occurrence of epilepsy and schizophrenia. In his monograph[3] published in 1918 he reports 4.6 per cent of schizophrenics observed in the Greifwald Clinic between 1909 and 1914 as experiencing convulsize seizures.

Jelliffe and White[4] think that convulsize seizures of epileptiform, but more often of hysteriform variety, may occur in dementia praecox, especially in the catatonic group.

Urstein,[5] who made a careful observation of epileptic attacks in dementia praecox, is inclined to look upon them as catatonic manifestations or insults. In a review of his casuistic material of 200 female and 180 male cases of dementia praecox, epileptiform seizures were observed by him in 19.5 per cent of the female and 8 per cent of the male patients. Summing up these studies, he states: "Taking it in all, I would like to add to the circular, paranoid, and hebephrenic form of catatonia a fourth form, the epileptic, and I have reason to believe that numerous inmates in the institutions for epileptics are such cases of catatonia."

Many others[6,7] feel that convulsions of the *grand mal* type do occur in schizophrenic episodes and definite cases of schizophrenia during the downward course of epilepsy.

Difficulties in Appraising the Therapeutic Results of Shock Therapy

A certain number of schizophrenic patients will improve however little may be done for them. A certain number will die on the wards. What has to be shown to permit an estimate of the value of any therapeutic measure is how much the average results have been improved, which is a difficult task in any research.[27] Dr. Roy G. Hoskins of the Harvard Medical School remarked, "Even in the case of the common cold which has had extensive study for a long while, we still do not know what measures are best or how good they are. If you contrast the relative simplicity of getting controls in such problems as that of the common cold with the complexities of research on psychiatric problems, you realize how far we are yet from an adequate appraisal of the value of the newer methodologies, indeed, we do not even know how good psychiatry is for psychotics."

To this is added the lack of unanimity in criteria for the diagnosis of schizophrenia, often resulting in the application of the term with scant supporting evidence. This has been due in part to obscure etiology and somatic pathology, in part to a feeling that the syndrome brought together a strange set of bedfellows only distantly related to one another if at all. Rosanoff reflects this chaotic situation in the 1938 edition of his text,[8] where he states that he has been "compelled by the nature of things" to omit schizophrenia as an entity.[29]

Another obscuring element in the evaluation of shock therapy in schizophrenia is the fact that the highest percentage of cures is reported in cases of less than six months duration where a faulty diagnosis is of frequent occurrence. In this connection, Langfeldt[9] shows that the psychoses designated as schizophrenia can be divided into two main groups: (1) the typical schizophrenias which in Kraepelin's terminology are designated as dementia praecox; (2) the so-called schizophrenias or "schizophreniform psychoses." This second group includes chiefly persons with manic-depressive features, and persons with psychopathic tendencies (paranoid, hysterical, asthenic, cyclothymic, schizoid, and others). The typical and atypical schizophrenias differ greatly in their prognosis. The atypical are nearly all spontaneously cured. and except for a few cases with an intermittent course, the cure is permanent. Shock treatment shortens the duration of these atypical cases; however, it cannot be definitely said as yet whether this is a permanent advantage. Langfeldt emphasizes that all reports on shock therapy of schizophrenia should give attention to the differentiation between typical and atypical cases.

The psychotherapeutic factor during and after metrazol treatment plays a very important role, adding to the difficulty of evaluating the effect of metrazol. How much is accomplished through personal attention to the patient, and how much through the use of metrazol *per se*, may ultimately have to be determined on a statistical basis.

With regard to the evaluation of the degree

of recovery or improvement, we must take into consideration the element of subjectivity of the examiner. The enthusiasm aroused by a new dramatic method of treatment and the active interest of the therapist in getting results after spending so much time and effort are factors which may account for some unintentional leniency in the estimation of the condition of patients receiving treatment.

Finally, a critical review of the results of shock therapy is made more difficult by the difference in terminology used by the members of the various hospital staffs, together with the different standards of criteria for "improvement" and "remission". The writers are therefore presenting in brief the criteria upon which they base their statistical data for remission (+++), social improvement (++), and institutional improvement (+). One may legitimately speak of a "cure" or "remission" when the patient is:

1) Judged by his physician to have regained his normal state.

2) Considered normal by his relatives and those about him.

3) Socially rehabilitated, i.e., when he resumes his previous occupation or adapts himself to work necessitating equal or superior initiative.

4) Free from relapse for several years.

The criteria for "social improvement" were the following:

1) Increased interest and contact.

2) Marked decrease in apathy and untidy habits.

3) Change in mental content and psychotic ideation.

4) Development of insight.

5) Improvement in conduct.

6) Social adaptation.

Patients unable to make a "social adaptation" were marked "institutional improvement" when the following were noted:

1) Less noisiness.
2) Less destructiveness.
3) Less untidiness.
4) Less uncooperativeness.
5) Less mutism.
6) Adjustment on a higher level in a better and quieter ward.
7) Working in occupational therapy or helping in the care of other patients.

Clinical Reactions, Complications and Hazards of the Treatment

It is not the intention of the writers to go into detailed description of the technique of treatment nor the mechanics of the convulsion, as this has already been adequately covered; they will confine themselves to the more significant items. They followed Meduna's original technique with slight individual modifications. A 20 or 18 gauge needle was used for the very rapid intravenous injections, for the determining factor in producing a convulsion is apparently the momentary concentration in the blood, not the absolute amount of the drug. Treatment began with 3 cc. of the 10 per cent solution. As a tolerance for the drug developed, the dosage was increased by 1 cc. when a convulsion was not obtained; however, no more than one dose was given a day.

As the psychotherapeutic factor and the ability of the medical personnel to establish rapport with the patient play a considerable part in the results of treatment, the drug was administered every other day, allowing the interim for psychotherapy. The convulsive seizure usually was found to follow within 10 to 40 seconds of the injection, although delayed action occurred in rare instances; one patient reacted 6 minutes following the injection. Theoretically, alkalinization of the patient should lower his convulsive threshold so that smaller dosage will be required to obtain a reaction. However, a group of 20 patients received sodium bicarbonate to the point of urine alkalinization without perceptible change in their convulsive threshold. A number of patients went through a period of depression, requiring close observation. The changes in the blood and urine were temporary, and are virtually the same as observed in epilepsy by numerous authors. The neutrophiles increased from 10 to 15 per cent with a proportional reduction of the lymphocytes. The acidity of the urine was increased about 20 per cent, ammonia about 12 per cent, phosphates about 10 per cent, and the chlorides decreased about 25 per cent.

Minor complications were encountered, such as general soreness, backache, nausea, vomiting, headache, biting of the tongue, and dislocations (especially of the jaw), all of which can be prevented by appropriate meas-

ures. Of the more serious complications, fractures were the most frequent. Unfortunately, a systematic x-ray check-up was conducted in only one of the buildings, and in 20 patients who reacted with no less than 20 convulsions each, six fractures were discovered: one fracture of both femoral necks and five compression fractures of the vertebrae, the latter being asymptomatic except for a backache of several days duration. Numerous fractures were reported by others, and intensive work is now being conducted to prevent their occurrence. Various orthopedic restraint devices such as hyperextension of the spine, preliminary insulin coma, and even spinal anesthesia along with metrazol, give no constant assurance that fractures can be prevented.[10] Bennett[11] claims that "aqueous or alcoholic extract of curare given parenterally in physiologic dosage sufficient to produce flaccid generalized motor paresis adequately protects the patient from traumatic complications of convulsive shock therapy. However, further experimentation would seem necessary before this procedure can be safely recommended for general psychiatric practice." Compression fractures in epilepsy and tetanus have been reported in the literature,[12] and the writers are at present conducting studies on compression fractures of the spine during convulsions of epilepsy, arteriosclerosis, and cerebrospinal syphilis; reports of these studies will be made elsewhere.

Rarefactions and bone lesions are considered by the writers to be contraindications to metrazol therapy. Metrazol therapy is also contraindicated in cardiovascular disease. Hayman and Brody[13] reported a case of death at the end of a metrazol convulsion, showing at autopsy a chronic endocarditis with acute cardiac dilatation. Von Angyal[14] reports death in a case with aortic disease. Goldstein[15] mentions the occurrence of cardiovascular collapse in one of his patients. Klein[16] reports paroxysmal tachycardia following metrazol convulsions. Cases of circulatory collapse due to arterial hypotension[17] have been reported. Great care must also be exercised in patients with initial hypertension, as the blood pressure in some of the cases here mentioned rose from 30 to over 100 mm. Hg. One patient had a latent pulmonary tuberculosis which became reactivated following the shock treatment, causing death several months later. The writers therefore consider incipient and latent tuberculosis contraindications to metrazol therapy. Among other contraindications are organic changes in the central nervous system, and acute or chronic infectious diseases. It is indeed fortunate that no fatalities were encountered during or immediately following the convulsions. Although fatal cases have been reported in the literature, Meduna[18] claims that metrazol therapy is accompanied by a mortality rate of only 0.22 per cent.

Possible Injury to the Central Nervous System

In animal experimentation there appears to be a great deal of evidence that the brain receives some damage. Miyashital[19] found changes in the brains of rabbits after metrazol convulsions, of the acute or subacute type of Nissl in the ganglion cells; these changes were diffuse, but involved all parts of the cortex. Liebert and Weil[20] found definite histopathologic changes in the brains of rabbits after metrazol convulsions. The intensity of the pathologic process roughly corresponded to the total amount of metrazol injected. According to Grayzel,[21] paroxysms in rabbits caused by insulin produce cerebral damage. When no convulsions followed insulin injections, there were no microscopic histopathologic changes in the brain; after slight convulsions there were minimal or no brain changes; after one or more serious convulsions there were definite anatomic lesions. The greater the number and the more prolonged the convulsions, the more severe were the lesions. What bearing these histopathologic observations have on convulsant therapy with metrazol can be only a matter of conjecture. As for definite histopathologic changes in the human brain, very little as yet can be stated; the method is comparatively new, and very little material has come to autopsy. Dr. Solomon[22] of the Boston Psychopathic Hospital reported a case showing, after metrazol treatment, definite neurological pathology which has been fixed and lasting, not fleeting. Hassin[23] reports a case which came to autopsy, in which swelling of the ganglion cells, chromatolysis and occasional edema, liquefaction and neuronphagia were the outstanding pathologic features. The grand mal seizure is a *(conditio) sine qua*

non of therapeutic efficiency of metrazol, and a convulsion is a dangerous procedure whenever it occurs. We do not know what is idiopathic epilepsy, but the convulsion of idiopathic epilepsy is regarded as the culmination of much more complex and deep seated disturbances. It is highly improbable that the convulsion artificially produced by metrazol duplicates this process.[28] In view of the observations and anatomical findings in animals, it can be concluded that the heart and the brain are the organs principally involved in the chemical disruption of metrazol. The convulsion is probably produced by anemia followed by hyperemia of the brain cortex and stem and possibly other parts of the nervous system. Metrazol therapy produces abnormal changes in the brain, even though these changes may be transitory. It is still doubtful, however, whether convulsions *per se* lead to definite organic cerebral lesions.

Therapeutic Results

It has long been known that shock may relieve mental illness. It is also known that severe illness may for a time favorably influence a case of schizophrenia or stop epileptic seizures. All these effects, however, are transitory. There is no doubt that metrazol produces changes in schizophrenic psychosis; whether these changes are always beneficial or not, and how lasting the benefits may be, are questions yet to be answered. In evaluating the results of metrazol shock therapy in schizophrenia, we are therefore concerned not so much with the immediate effects of the treatment as with its lasting effect, the permanency or duration of the improvement. The present report is based on the treatment of a group of 82 patients, equally divided by sex, ranging in age from 18 to 52, the average being 31, the duration of their illness ranging from 1 to 20 years, the average being 4½. The various types of schizophrenia were equally represented. Chart No. I is based on the condition of the patient immediately following the treatment and after a follow-up study ranging from 6 months to 2 years following termination of treatment. Chart No. II is based on the condition of a group of 212 unselected patients with schizophrenia admitted to the hospital within the same two year period as the group in Chart No. I; this latter group was not subjected to shock therapy.

CHART NO. I

AFTER SHOCK THERAPY

	Immediately after treatment		*After follow-up study*	
Degree of improvement	*No. of Patients*	*Per Cent*	*No. of Patients*	*Per Cent*
O No improvement	41	50	63	76
+ Hospital improvement	26	32	8	10
++ Social improvement	15	18	11	14
+++ Remission	—	—	—	—

CHART NO. II

WITHOUT SHOCK THERAPY

Degree of improvement	*No. of Patients*	*Per Cent*
O No improvement	143	67
+ Hospital improvement	59	28
++ Social improvement	10	5
+++ Remission	—	—

To summarize the therapeutic results:

Immediately following completion of treatment, 50 per cent of the patients were improved; 32 per cent were hospital improvements, and 18 per cent were socially improved; there were no complete remissions.

After a follow-up study from six months to two years after termination of treatment, only 24 per cent of the patients improved; 10 per cent were hospital improvements; 14 per cent were social improvements, and there were no complete remissions. One notes, therefore, a relapse of 50 per cent of the patients who showed original improvement.

In the group not receiving shock therapy a greater percentage is noted of non-improvements, a lesser percentage of hospital improvements, and a still lesser percentage of social improvements, with no remissions.

A more detailed analysis revealed:

The percentage of improvements in the male group was slightly higher than in the female. As to types of schizophrenia, the difference was negligible.

Both "treated" and untreated patients with psychosis of shorter duration had the more permanent improvements.

The highest percentage of relapses occurred among patients with psychosis of longer duration.

Patients with psychosis of shorter duration showed the highest percentage of improvements.

The higher the degree of improvement following the treatment, the lower the percentage of relapses.

Patients with a higher percentage of convulsive seizures in the series of injections showed better results than those with incomplete reactions such as aura, confusion, furor, and other equivalents.

There was an average gain in weight of about five pounds.

Fear and submission were prominent symptoms in some of the patients, and one of the authors (S. A.—24) utilized these phenomena in a group of patients with prolonged refusal of food, necessitating tube-feeding for long periods. The patients resumed eating under metrazol therapy.

One of these patients showed only hospital improvement. Three months after shock therapy he developed a cholesystitis; the stones were removed and the gall bladder drained. After operation, the patient made a rapid social improvement and is now at home socially adjusted eight months after the operation.

Discussion and Conclusions

The shock treatment of psychoses goes on with somewhat diminished enthusiasm. Convulsive shock therapy has been enthusiastically endorsed by many workers and critically condemned by others. The former maintain that much of the personal discomfort of our mental patients, the burden of care to the hospital, and their economic inefficiency are due to such traits as uncooperativeness, noisiness, destructiveness, and untidiness. If a method of treatment has been found that will overcome these traits, it might be well worth adopting. The other extremists[25] maintain that "one feels doubtful of the ethical standards that allow shock treatment to be given for the purpose of making nursing easier for the hospital administration. It comes dangerously close to punishment." There is much skepticism and an insistence that the original claims of European workers of "80 per cent remissions" were statistically unsound. If we consider 50 per cent improvement immediately following the treatment in our group of chronic cases, it may be admitted that in a group of highly selected cases of not over six months duration, the improvements immediately following the treatment may reach higher figures. However, a follow-up study of these so-called remissions is now indicated. The better controlled results in this country indicate that the shock treatment in cases of early schizophrenia causes remissions sooner than could be expected with general hospital care. In patients with chronic disease the shock treatment very often improves their mental status so that they can be moved from a disturbed ward to a better environment and become useful in the hospital, but less often do remissions occur that allow patients to make a successful adjustment at home. Follow-up studies by the writers six months to two years following the treatment are not very encouraging, since relapses are frequent. For that reason the exact status of metrazol as a permanent therapeutic agent in neuropsychiatry is yet to be determined. It must not be overlooked that the creation of a situation for the patient in which he is rendered helpless and needs the help of the physician and personnel may result in making the patient more receptive to other forms of therapy. He should be given the benefit of an early trial at home, as relapses occur frequently if such trial is delayed. All emphasis is directed toward showing the patient that something has transpired to make him different and that now he is well enough for a visit at home or to make good. In many instances success is achieved with metrazol when the usual hospital treatment has failed. It would, therefore, be unwise to entirely abandon the metrazol therapy; however, it is also unwise to use it indiscriminately as a panacea for all cases of schizophrenia. It is drastic treatment, and the drug should be handled only with great respect and in cooperative patients after a thorough physical examination, routine laboratory studies of the urine and blood (including blood sugar and NPN), x-ray examination of the chest, electrocardiograms, and a basal metabolic test.

Summary

1) A brief review is presented of the literature on the various phases of metrazol convulsive shock therapy in schizophrenia.

2) A group of 82 patients of the chronic

type of schizophrenia, equally divided by sex, underwent a course of metrazol therapy. The results immediately following the treatment are compared with their condition after a follow-up study of six months to two years. The results in this group of metrazol-treated patients are contrasted with the results obtained in a group of 212 patients who did not receive shock therapy.

Spring Grove State Hospital.

References

1 Von Meduna, L.: "New Methods of Medical Treatment of Schizophrenia," *Arch. Neurol. and Psychiat., 35:* 361-363 (Feb.) 1936.
2 Kraepelin, E.: "Zur Epilepsiefrage," *Ztschr. f. d. ges. Neurol. u. Psychiat., 52:* 107, 1919.
3 Vorkastner, W.: *Epilepsie und Dementia Praecox.*, Berlin: Karger, 1918.
4 Jelliffe, S. E. and White, W. A.: *Diseases of the Nervous System,* Lea and Febiger, Fifth Edit., 1929.
5 Urstein, M.: *Mannisch-Depressives Irresein und Periodisches Irresein abs Erscheinungsform der Katatonie.*, Berlin: Urban u Schwarzenberg, 1912.
6 Faver, Harry E.: "Convulsions in Schizophrenia," *Psychiatric Quarterly, 12:* 725, 1938
7 Murphy, John P. H.: "A Comparative Study of Epilepsy and Schizophrenia," *Med. Journ. and Rec., 128:* 77 and 110, 1928.
8 Rosanoff, Aaron J.: *Manual of Psychiatry and Mental Hygiene,* 7th Ed., 1938.
9 Langfeldt, G.: "Question of Spontaneous Remissions of Schizophrenic Psychoses with Special Consideration of Duration of These Remissions: What Can Be Obtained with the Shock Therapy of These Conditions?" *Zeitschrift f. d. ges. Neurol. u Psychiatrie.*, Berlin: 164-494 (Jan. 23) 1939.
10 Hamsa, W. R. and Bennett, A. E.: "Traumatic Complications of Convulsive Shock Therapy: A Method of Preventing Fractures of the Spine and Lower Extremities," *J. A. M. A., 112:* 2244-2246 (June 3) 1939.
11 Bennett, A. E.: "Preventing Traumatic Complications in Convulsive Shock Therapy by Curare," *J. A. M. A., 114:* 322-324 (Jan. 27) 1940.
12 Ziskind, Eugene and Somerfeld - Ziskind, Esther: "Compression Fractures of the Spine in Epilepsy," *Bull. Los Angeles Neuro. Soc., 4:* 45 (Mar.) 1939.
13 Hayman, Max and Brody; Morris, W.: "Metrazol Therapy in Schizophrenia," *J. A. M. A., 112:* 310 (Jan. 28) 1939.
14 Von Angyal, L. and Gyárfós, K.: "Ueber die Cardiazolkrampfbehandlung der Schizophrenie," *Arch. f. Psychiat., 106:* 1, 1936.
15 Goldstein, H. H.; Dombrowski, E. F.; Edlin, J. V.; Bay, A. P.; McCorry, C. L. and Weinberg, J.: "Treatment of Psychoses with Pentamethylentetrazol," *Am. J. Psychiat., 94:* 1347 (May) 1938.
16 Klein, F.: "Paroxysmal Tachycardia brought on by Pentamethylentetrazol (Corvis)," *Underl. Tydschr. v. Geneesk,* Amsterdam, *83:* 5049 (Oct. 21) 1939.
17 Adler, Alexander and McAdoo, Hosea W.: "Metrazol Reaction in Patients with Arterial Hypotension," *The Am. J. of Psych., 96:* 699 (Nov.) 1939.
18 de Meduna De Ladislas: "La Thérapeutique de la Schizophrénie par les Convulsions (Compte Rendu)," *J. belge de Neurol. et de Psychiat.*, 1939, 39 Annee: No. 4 (April) pp. 227-237.
19 Miyashital, K.: "Pathologic Changes Produced by Metrazol," *Psychiat. et Neurol.*, Japon. *43:* 2 (Jan.) 1939.
20 Liebert, Erich and Weil, Arthur: 'Histopathologic Changes in the Brain Following Experimental Injections of Metrazol," *Arch. of Neurol. and Psych.*, Vol. 42, pp. 690-699 (Oct.) 1939.
21 Grayzel, David M.: "Changes in the Central Nervous System Resulting from Convulsions due to Hyperinsulinism," *Arch. Int. Med., 54:* 694 (Nov.) 1934.
22 Solomon, Harry C.: Discussion at Worcester State Hospital in a Symposium on therapy, May 18, 1938.
23 Hassin, George B.: 'Cerebral Changes in Fatal Cases Following Treatment with Barbital U.S.P., Insulin, and Metrazol," *Arch. Neur. and Psych., 42:* 679 (Oct.) 1939.
24 Androp, Serge: "Metrazol Therapy in Cases of Prolonged Refusal of Food Complicating the Psychoses," Accepted for publication in the *Am. J. of Psych.*
25 Cobb, Stanley: "Review of Neuropsychiatry for 1939," *Archives of Internal Medicine, 64:* 1328 (Dec.) 1939.
26 Michelson, Harry: "The Use of Metrazol in the Treatment of Schizophrenia," *Symposium on Therapy,* published by the Massachusetts Dept. of Mental Health, Sept. 1939.
27 Hoskins, Roy G.: *Ibid.*
28 Rotchild, D.: *Ibid.*
29. Flower, B. H. and Cohen, Benjamin: *Ibid.*

(Continued from page 211)

case funds were not lacking to finance the defense. Subsequently, and because of his prominence, the prosecution invited the same psychiatrist to serve on its side. It is reported that he also declined this offer, saying that he would be glad to serve without charge and would pay his own expenses, if permitted to examine the accused impartially and scientifically and as a friend of the court.

This is not only a problem for the psychiatrist but for all doctors. Every time a physician is called as an expert witness, he may be placed in the embarrassing, and indeed unethical, position of testifying without having access to all of the facts. After all, no physician would think of treating a patient without examining him.

The battle of the experts must end. And it is up to organized medicine to remove this cloud, which to the general public, appears to cast a shadow upon the reputation of the medical profession.

P. L. S.

Neurological Complications of Sulfanilamide and Sulfanilamide Compounds*

A Review and a Report of Two Cases of Polyneuritis

HARRISON EVANS, M.D. *and* FERDINAND WELEBIR, M.D.
Columbus, Ohio *Los Angeles, California*

Dr. Evans received his medical degree from the College of Medical Evangelists in 1936. He is Neurologist for the University and Saint Francis Hospitals; Clinical Director of the Harding Sanitarium; and Instructor in Neuropsychiatry at the Ohio State University College of Medicine. Dr. Evans is a Diplomate of the American Board of Psychiatry and Neurology.

Dr. Welebir was graduated from the College of Medical Evangelists in 1937. He is Senior Resident in Urology and Genito-Urinary Surgery at the White Memorial Hospital, and Assistant in Urology at the College of Medical Evangelists in Los Angeles. Dr. Welebir is a Diplomate of the National Board of Medical Examiners and a Junior Candidate for the American College of Surgeons.

The use of sulfanilamide and related compounds in different infections is becoming increasingly widespread. The efficacy and even specificity of these drugs for many diseases which were formerly difficult if not impossible to treat successfully have brought added attention to them. For this reason, physicians frequently turn to them as drugs of primary use in many serious illnesses. Undoubtedly in most instances this is justified, but still judgment and caution are indicated. Complications from their use do arise, many of which are serious to the extent of being fatal, and this eventuality should deter any hurried or promiscuous use of these drugs for an illness which may not in itself carry a serious prognosis.

Many of the complications such as anemia, agranulocytosis, sulfhemoglobinemia, methemoglobinemia, leukopenia, skin rash, diarrhea, dizziness, tinnitus, and cyanosis are well known. But serious complications referable to the nervous system are not as well known, probably because they are not so frequent. It is our purpose to review the neurological complications consequent to the use of these drugs and to report two cases of polyneuritis which occurred following the use of sulfanilamide and Disulon (a disulfanilamide preparation).

* From the Departments of Neurology and Urology of the College of Medical Evangelists, Los Angeles, California.

Case Reports

Case I. Acute gonorrheal urethritis. Treatment with twenty grams of Disulon and eight grams of sulfanilamide in fourteen days. Motor polyneuritis of lower extremities six days after stopping treatment. No improvement in first month.

(White Memorial Clinic 132-660) Mr. P. E., White male, age 77, presented himself to the Genito-Urinary Clinic of the College of Medical Evangelists on November 22, 1939, with complaints of burning on urination and a urethral discharge of four weeks duration. A diagnosis of gonococcal urethritis was made after a smear of the urethral discharge showed characteristic organisms and the patient was placed on Disulon therapy on December 12, 1939. Between this time and December 22, he received sixty tablets, a total of twenty grams. He then received twenty-four tablets of sulfanilamide (8 grams) between December 22 and December 26, at which time medication was stopped.

Six days later, on arising in the morning, he noticed that his feet were weak. He had considerable pain and tenderness in the back of the legs. There were no other symptoms. He was examined on January 3, 1940, seven days after his last treatment and one day after the appearance of his symptoms. The examination revealed a bilateral complete foot drop, inability to abduct or adduct the toes, and moderate tenderness on deep palpation along the nerve trunks in the calves. There was no perceptible atrophy, and the strength of the thighs and upper extremities was not grossly altered. Sensation, except for vibration, was normal in all modalities. Vibration was absent to the level of the eighth thoracic segment. The knee jerks were diminished and the ankle jerks were absent. There was no plantar response. The cranial nerves were quite normal.

His progress was not encouraging. He re-

ceived vitamin B_1 in fairly large doses, both by mouth and intravenously, but when last seen, a month following the onset of the neurological symptoms, there was no material change from the condition present at the time of the first examination.

Case 2. Streptococcus pharyngitis. Treatment with sulfanilamide. Motor and sensory polyneuritis on fourth day. Treatment with vitamin B_1. Recovery in three weeks.

Mrs. Maria H., a Mexican female, age 34, who is being reported through the courtesy of Dr. Kenneth Abbott of the Abbott Clinic, Ontario, California, was treated with sulfanilamide for a streptococcus pharyngitis. She received thirty grains (2 grams) of sulfanilamide as the first dose and then fifteen grains (1 gram) every four hours until the fourth day when she began to complain of paresthesias of the fingers and toes.

Examination revealed moderate tenderness along the course of the sciatic nerve and also, but to a less extent, along the nerve trunks in the upper extremities. Motor weakness was minimal, limited to a slight foot drop on the left side. There was an hypesthesia to all sensation in the distal portions of both the upper and lower extremities, that in the hands being more definite along the distribution of the median and ulnar nerves. Sulfanilamide was stopped. Large doses of vitamin B_1 were given. Gradual improvement ensued over a period of three weeks, at the end of which time practically all symptoms had disappeared.

Review of the Literature

Only three cases of polyneuritis complicating sulfanilamide therapy have been reported, one case each by Ornsteen and Furst,[1] Janet[2] and Waugh.[3] However, many cases have been reported consequent to the use of di-sulfanilamide preparations. Such preparations are Diseptal and Uliron, which are a brand of di-methyl di-sulfanilamide and sulfanilyl-sulfanilamide, which is a di-sulfanilamide.[4,5] Dollken[6] and Grunig[7] collected between them thirty-six cases from the literature, which were due to Uliron, and added five cases each of their own which made a total of forty-six cases. We have reviewed approximately forty more cases[8,9,10,11,12,13,14,15,16,17] and undoubtedly there are others which have been overlooked.

The usual characteristics of the polyneuritis are as follows: It is predominantly motor with little if any sensory involvement other than spontaneous pain at the onset. The feet and legs are the sites of predilection. Foot drop, with involvement of the small muscles of the feet, indicating peroneal and tibial nerve involvement, is the characteristic and usual finding. The onset may be as early as twenty-four hours after the beginning of treatment, or delayed as long as eight to fourteen days after stopping treatment. The symptoms have persisted as long as a year. The average period is from two to five months. Recovery in some cases has been incomplete even after a year. Sensory symptoms in both the feet and hands have been reported by Ornsteen and Furst,[1] Van Valkenburg and Kreuzwendedich,[18] Kuhnau[19] and Wigton and Johnson.[20] These have appeared in the form of spontaneous pain initiating the side reactions and a dulling of sensation of a peripheral nerve distribution. Motor weakness in the hands is rare. Wigton and Johnson,[20] Grunig,[7] Kranz,[21] and Rost[22] reported instances in which gross weakness appeared with atrophy of the small muscles of the hands. Neuritis of the brachial plexus following the administration of 31.5 grams of Uliron has been reported by Fischer and Grunig.[7] Electrical changes depend somewhat on the seriousness of the involvement. In some cases there have been no changes, but in others reaction of degeneration has occurred.

The most serious complication reported has been a myelitis or an encephalomyelitis. Fisher and Gilmour[23] reported two cases of encephalomyelitis following the use of sulfanilamide, one of which ended fatally. Santo[24] and Schubert[25] each reported a fatal case of myelitis following Uliron therapy. Roch's and Neeser's[26] patient presented signs of an amyotrophic-lateral sclerosis after disulfanilamide therapy in which recovery took place. Lilienthal[27] also reported a case of myelitis following Uliron therapy with recovery.

Fisher's and Gilmour's[23] fatal case received .5 grams of sulfanilamide twice daily for rheumatoid arthritis and lupus erythematosis. Cyanosis appeared the second day. The thirteenth day a fever of three degrees with a slight headache appeared. On the fifteenth day a flaccid paralysis of the legs developed.

Muscle rigidity and absent knee, ankle, abdominal and plantar reflexes were noted. Position sense of the toes was lost and touch was absent to the iliac crest while pain and vibration were still perceived. The spinal fluid was turbid with 16,000 cells, mainly polymorphonuclears, and the spinal fluid pressure was elevated. The protein was 100 mgms. per cent. On the sixteenth day sensation was lost to the fifth dorsal segment. Retention of urine developed. Death occurred on the eighteenth day. Perivascular demyelination and inflammatory changes in the vessel walls were found. Other vascular changes such as petechial hemorrhages, thrombosis of the arteries and veins, especially in the leptomeninges and also in the cord, were noted.

Their second case, a twenty-eight year old woman, received one gram of sulfanilamide three times daily for six days for sore throat, rheumatic complaints and erythema nodosum. Cyanosis was present the first twenty-four hours. Six days later the temperature rose to 103°. Severe headache, vomiting and shooting pains in the right leg were present. The knee and ankle jerks were exaggerated and ankle clonus was present. The spinal puncture was negative. The temperature became normal four days after stopping the sulfanilamide and at the time of discharge the right leg was still somewhat weak and the reflexes were about the same as before.

Roch's and Neeser's[26] case, which presented a picture of amyotrophic-lateral sclerosis was given thirty-five grains of di-sulfanilamide preparation in approximately fifteen days. Five days after the treatment was stopped, atrophy of the interossei, thenar and hypothenar muscles was evident. There was reaction of degeneration of the muscles of the thenar and hypothenar eminences. The lower limbs were paralyzed. The deep reflexes were increased in the legs and ankle clonus was present but no Babinski sign was found. No sensory changes, objectively or subjectively were evident. Improvement occurred within eighteen days and within five weeks the patient was able to leave the hospital.

Santo's[24] and Schubert's[25] cases of myelitis following Uliron therapy ended fatally. Postmortem studies were made. Gross degeneration and massive softening of the cord were found. Lilienthal's[27] case was not fatal but clinically there were signs of myelitis. There was spasticity of the legs. The knee and ankle jerks were active, the right greater than the left. There were bilateral Oppenheim and Gordon signs.

The cranial nerves have been involved in some instances. Bucy[28] reported a case of optic neuritis after the use of sulfanilamide in small doses. The patient was given sulfanilamide on three occasions. Seven and a half grams were given in the first course, but treatment was stopped for a short period because of cyanosis, diarrhea and a choking sensation. The second time 6.6 grams were given, but discontinued because of a recurrence of previous symptoms. Again after a short interval .3 of a gram was given and optic neuritis accompanied by a marked loss of vision appeared.

Van Valkenburg and Kreuzwendedich[18] had a patient who showed dysesthesia in the entire area of the fifth nerves. Also the pharyngeal muscles were weakened so that swallowing was difficult and the sense of taste was lost following Uliron therapy.

Myopia has occurred in several instances following the use of sulfanilamide, sulfapyridine and another sulfanilamide compound.[29,30,31,32] It is not usually of long duration—not more than several days. The cause of the myopia is not known definitely. Heinonen[31] suggests it is due to an irritation of the parasympathetic fibers to the ciliary muscles. Spellberg[29] thinks, on the other hand, that it is due to edema of the retina.

Sulfanilamide has apparently been the precipitating factor in the development of a delirious or psychotic state on at least three occasions.[33,34,35] In two instances the patients presented a disturbed confusional state following the administration of a single dose.[33,34] This occurred on repeated trials. The third patient developed a paranoid reaction which persisted for a prolonged period of time.[35]

Discussion

Neurological complications are apparently due to several factors. It is evident that di-sulfanilamide preparations are more prone to cause neuritis than are others, for practically all of the cases reported have resulted from the use of Uliron or some other brand of di-sulfanilamide. The amount of the drug given is also important according to

Dollken[6] and Grunig,[7] for they have found that too large a dose or prolonged treatment is more prone to cause a neuritis. Deakin's[36] experience would tend to bear this out. He has treated two hundred and fifty-six patients with Uliron preparations (the sodium salt of Uliron and the Uliron base) with forty grains daily for four days followed by a weeks rest before another course is given. He has had no serious side reactions. And the minor side reactions were noticeably less than with sulfanilamide which was given continuously without a rest period. Thus it seems that the variability in the toxicity of the drug and the amount of the drug used are important. However there seems also to be an individual susceptibility. For example, Bucy's[28] patient developed toxic manifestations on three different occasions after relatively small doses—the third time after only .3 gram of sulfanilamide. Grunig[7] reported a case of polyneuritis developing after only 3 grams of Diseptal C (a brand of di-sulfanilamide). A confusional, irrational and disoriented episode followed one dose of sulfanilamide on repeated occasions.[33,34] It is the opinion of some that side reactions are due entirely to an idiosyncrasy to the drug.[37] The patient's physical status, amount of exercise and habits are supposed to play some part. McGinty[38] and Dollken[6] both state that neuritis is more apt to develop in individuals who are ambulatory and especially if doing any great amount of walking. Certain experimental work with pigeons by Engelhardt and Birkenmaier[39] and Engelhardt and Hullstrung[40] tend to confirm this. Dollken[6] and Kranz[21] advise against the use of alcohol while under treatment and Kranz[21] even advises against smoking. Vitamin B_1 is thought to play a part by some and nearly all have given it when a neuritis developed. Certain observers[40] have suggested that the drug causes a gastritis with impaired absorption of vitamin B_1 which in turn is at least partially responsible for the neuritis. These same observers found that by administering vitamin B_1 to pigeons, even though they were exercising to a great extent, a neuritis did not develop. Rosenthal[41] in experiments with rabbits found there was some relation of diet to the toxicity of sulfanilamide but the explanation awaits further work. Bucy[28] and Paton and Eaton[42] caution against the use of other sulfur preparations shortly before or simultaneously with the use of sulfanilamide for thereby its toxicity is likely to be enhanced. Bucy's[28] patient had received ferrous sulphate at the same time the sulfanilamide had been administered.

Summary

In summarizing, a review of the literature shows that sulfanilamide and more often its derivatives may, and not infrequently do, cause neurological complications of severe degree. These untoward reactions may affect various parts of the nervous system, including the central nervous system, the cranial nerves and the peripheral nerves, the latter being most frequently involved. The precise cause of these complications is not definitely known, but it seems likely that it is due to the relative toxicity of the drug as well as the amount of the drug used. However, an individual susceptibility and idiosyncrasy apparently must be acting for these complications to occur. This individual susceptibility may be somewhat dependent on the person's physical condition, the state of nutrition, particularly the amount of vitamin B_1 received and the degree of physical activity. Other sulfur compounds should not be used prior to or simultaneous with the use of sulfanilamide.

References

1 Ornsteen, A. M. and Furst, W.: "Peripheral Neuritis due to Sulfanilamide," *J.A.M.A., 111:* 2103 (Dec. 3) 1938.
2 Janet, J.: "Protest Against Sale of Sulfanilamide over the Counter," *J.A.M.A., 110:* 1501 (April 30) 1938.
3 Waugh, J. R.: "Peripheral Neuritis during Administration of Sulfanilamide," *Am. J. Syph., Gono., and Ven. Dis., 23:* 745 (Nov.) 1939.
4 Bigler, J. A. and Haralambie, J. Q.: "Sulfanilamide and Related Compounds. A review of the Literature," *Am. J. Dis. Child., 57:* 1110 (May) 1939.
5 Bauer, H. and Rosenthal, S. M.: "Studies in Chemotherapy VII. Some New Sulphur Compounds Active Against Bacterial Infection," *Pub. Health Rep., 53:* 2, 40 (Jan. 14) 1938.
6 Dollken, H.: "Nebenwirkung des Ulirons dei der Gonorrhoe Behandlung," *Dermat. Wchnschr., 107:* 1273 (Oct. 29) 1938.
7 Grunig, E.: "Schadigungen bei der chemotherapeutischen Behandlung der Gonorrhoe mit Diseptal," *Med. Klin., 34:* 1399 (Oct. 21) 1938.
8 Hullstrung, H. and Krause, F.: "Polyneuritis nach Sulfonamidhaltigen Verbindungen bein Menschen und Tauben," *Deutsche med. Wchnschr., 64:* 114 (Jan. 21) 1938.
9 Hinzpeter, H.: "Uliron and Ulironneuritis," *Zentralbl. f. Gynak., 62:* 2052 (Sept. 10) 1938.
10 Swiatkiewicz, J.: "Polyneuritis i Polyneu-

romyositis Joke Nastepstwo Leczniczego Stosowania Ulironu Preciurzezaczce," *Polska gaz. lek., 9:* (Feb. 26) 1939.
11 Rojas, L.: "Un Caso De Polineuritis Producida Por El 'Uliron,'" *Actas dermo-sif., 30:* 221 (Jan.) 1939.
12 Gelbjerg-Hansen, G. and Krabbe, K. H.: "Polyneuritis Fremkaldt Af Uliron," *Ugesk. f. laeger., 100:* 568 (May 19) 1938.
13 Bruns-Pedersen, M. and Dalsgaard-Nielsen, T.: "Neuritis Ved Ulironbehandling," *Ugesk f. Laeger., 100:* 590 (May 26) 1938, (Quoted by Waugh).
14 Shelley, H. S.: "A Report on the Treatment of Gonococcal Urethritis with Sulfanilyl-Sulfanilamide," *Bull. School Med., Univ. Maryland, 23:* 3, 123 (Jan.) 1939. (Quoted by Waugh).
15 O'Crowley, C. R.; James, W. L. and Sutton, H. L.: "Sulfanilyl-Sulfanilamide in the Treatment of Gonorrhea in the Male," *J. Urol., 41:* 1, 51 (Jan.) 1939. (Quoted by Waugh).
16 Smith, R. S.: "A Toxic Neurological Syndrome due to Di-sulfanilamide," *Hosp. News, U.S.P. H.S., 5:* 12, 1938. (Quoted by Waugh).
17 Bannick, E. G.; Brown, A. E. and Foster, F. P.: "Therapeutic Effectiveness and Toxicity of Sulfanilamide and Several Related Compounds: Further Clinical Observations," *J.A. M.A., 111:* 770 (Aug. 27) 1938.
18 Van Valkenburg, C. T. and Kreuzwendedich von dem Borne, G. A.: "Polyneuritis after Uliron Treatment," *Lancet, 2:* 889 (Oct. 15) 1938.
19 Kuhnau, W. W.: "Beitrage zur Klinik der Diseptalneuritis, ihrer Entstehung und Verhutung," *Med. Klin., 35:* 377 (March 24) 1939.
20 Wigton, R. S. and Johnson, S. H. III.: "Peripheral Neuritis Following Sulfanilyl-Sulfanilamide (disulfanilamide), Report of Four Cases," *J.A.M.A., 111:* 1641 (Oct. 29) 1938.
21 Kranz, H.: "Ulironschaden des Nervensystems," *Ztschr. f. d. ges. Neurol. und Psychiat., 165:* 269-272, 1939.
22 Rost, J.: "Polyneuritis nach peroraler Verabreichung Sulfanamidhaltiger Arzneimittel," *Monatschr. f. Psychiat. u. Neurol., 100:* 92 (Sept.) 1938.
23 Fisher, J. H. and Gilmour, J. R.: "Encephalomyelitis Following Administration of Sulphanilamide, With Note on Histological Findings," *Lancet, 2:* 301 (Aug. 5) 1939.
24 Santo, E.: "Uber eine Schwere Erkrankung des Ruckenmarkes nach Ulironbehandlung einer Gonorrhoe," *Frakfurt. Ztschr. f. Path., 53:* 105-119, 1939.
25 Schubert, M.: "Weitere Erfahrungen mit der Chemotherapie der Gonorrhoe," *Dermat Wchnschr., 107:* 807 (July 2) 1938.
26 Roch, M. E. and Neeser, J.: "Syndrome Neurologique, Simulant la Sclerose Laterale Amyotrophique Provoque par un Medicament Sulfamide (4, 4 Diaminophenyl-Sulfanilanilamide)," *Bull. et. Mem. Soc. Med. d. hop. de Paris, 55:* 885 (June 12) 1939.
27 Lilienthal, W.: "Die Behandlung der Mannlichen und Weiblichen Gonorrhoe mit den Chemotherapeutika Diseptal (Uliron) und Diseptal B-1," *Dermat. Wchnschr., 107:* 833 (July 9) 1938.
28 Bucy, P. C.: "Toxic Optic Neuritis Resulting from Sulfanilamide," *J.A.M.A., 109:* 1007 (Sept. 25) 1937.
29 Spellberg, M. A.: "Toxicity of Sulfanilamide. Severe Transient Myopia Following Use of Sulfanilamide, Report of case," *Ill. M. J., 75:* 366 (April) 1939.
30 Fange, G.: "Ett Fall av Transitorisk Myopi Upptradande i Anslutning Till M & B 693 Behandlung av Pneumoni," *Svenska lak.-tidning, 36:* 1197 (June 9) 1939.
31 Heinonen, O.: "Sulfonamidi Transitorism Myopian Todennakorsena Aihenttajana," *Duodecim, 55:* 552-556, 1939.
32 Langrange, H. and Laudat, M.: "Myopie Spasmodique Transitoire, Accident de la Medication Organique Soufree," *Bull. et mem. soc. med. d. hop. de. Paris, 55:* 883 (June 12) 1939.
33 Danziger, L.: "Delayed Toxic Reaction to Sulfanilamide," *Bull. Johns Hopkins Hosp., 63:* 340 (Nov.) 1938.
34 Garvin, C. F.: "Complications Following the Administration of Sulfanilamide," *J.A.M.A., 113:* 288 (July 22) 1939.
35 Hogan, B. W. and McNamara, P. J.: "Psychosis Precipitated by Sulfanilamide," *U. S. Nav. M. Bull., 36:* 60 (Jan.) 1938.
36 Deakin, R.: *Personal Communication.*
37 Wiseman, B. K.: "In Discussion to Garvin's Paper," *J.A.M.A., 113:* 288 (July 22) 1939.
38 McGinty, A. P.: "A Review of the Complication Following the Administration of Sulfanilamide," *J.M.A. Georgia, 27:* 21, 1938.
39 Engelhardt, W. and Birkenmaier, O: "Lahmungserscheinungen bei Tauben nach Verabreichung Sulfonamidhaltiger Verbindungen und Zusatzlicher Muskelarbeit," *Klin. Wchnschr., 17:* 1325 (Sept. 17) 1938.
40 Engelhardt, W. and Hullstrung, H.: "Die Verhutung Lahmungsahnlicher Erscheinungen bei Ulirongefutterten Tauben Durch Vitamin B-1," *Klin. Wchnschr., 18:* 774 (June 3) 1939.
41 Rosenthal, S. M.: "Studies in Chemotherapy VIII. Some Toxic Effects of Repeated Administration of Sulfanilamide and Sulfanilyl-Sulfanilamide (Di-sulfanilamide) to Rabbits and Chickens," *Pub. Health Rep., 54:* 4, 95 (Jan. 27) 1939.

Your Comments on this Journal will be Appreciated

We would also be pleased to know of any subjects you want covered.

Write to

PHYSICIANS POSTGRADUATE PRESS

500 North Dearborn Street, Chicago, Illinois

The Incidence of Contributory Factors and Associated Conditions in Vascular Encephalopathy*

CULLEN WARD IRISH, M.D.
Los Angeles, California

Dr. Cullen Ward Irish was graduated from the Ohio State University, College of Medicine at Columbus, in 1915. He is an Associate Professor of Neurology and Psychiatry at the College of Medical Evangelists at Los Angeles. He is a Fellow of the American College of Physicians, and a member of the American Psychiatric Association. He has contributed many valuable papers to the medical literature.

In a clinical pathological survey of 1,000 cases of vascular encephalopathy found at 12,503 consecutive necropsies, done at the Los Angeles General Hospital between March, 1918, and November, 1934, an investigation was made of the incidence of the contributory factors and associated conditions present. The causative factors productive of embolic processes and sinus thromboses are usually of obvious infectious origin, though mechanical agencies may result in embolism.

In the 133 instances of *cerebral embolic* processes, the origin of the lethal embolus was cardiovascular in 78.9 per cent, from other sites in 16 per cent and from undetermined sources in the remainder, 5.1 per cent. The emboli were of infectious origin in 119 (88%) and likely entirely mechanical in 8 (6%).** The incidence of various contributory conditions in the production of sinus thromboses in the 98 cases with that type of involvement is indicated in the accompanying chart.***

Thrombotic and hemorrhagic lesions follow a number of provocative pathologic and physiologic processes, many of which are common to both types, though demonstrating varying mechanisms and incidences.

The comparative role of the various associated conditions in the production of the cerebral vascular insult is often impossible to evaluate. No accurate estimation of the causative attributes can be made in all instances but investigation of their incidences is of much informative value. An embolus brought by the blood stream from another source and lodged in a cerebral vessel is usually of infectious origin, most frequently from cardiac valve vegetations, or may originate as loosened pieces of thrombi and arterio-sclerotic plaques in other vessels. Compression of a vessel supplying the cranial contents may also result in an embolic occlusion of a cerebral vessel.

In thrombotic and hemorrhagic cerebral vascular conditions many more provocative factors present themselves but a number of obvious influences and some directly causative of cerebral pathology may be briefly delineated. In general, a number of endogenous and exogenous factors, similar in character, may play a part in the production of either cerebral thrombosis or cerebral hemorrhage. Closer study of these factors discloses that some specific conditions are more liable to result in one or the other—infections or toxic conditions and blood dyscrasias being more frequently productive of hemorrhage, and chronic debilitating diseases being more prone to be followed by thrombotic cerebral lesions.

Cerebral arteriosclerosis is, undoubtedly, directly influential in the production of either type of lesion. The vascular wall becomes sufficiently weakened by this process so that it is scarcely able to withstand normal vascular tension and readily ruptures with increased intra-vascular pressure. Or, roughening of its intima and restriction of its lumen causes slowing of the blood stream at the involved site and promotes the occurrence of thrombosis. It has been demonstrated that a normal vessel will withstand several times the pressure it is ever called upon to meet in any condition imposed, hence a vascular pathological process is always necessary for its rupture to occur. Likewise, it is questionable whether or not a thrombotic lesion can result without local involvement of the vessel occluded. It is conceivable that abnormal blood states due to chemicals or radical changes in

* Read before the Neuropsychiatry Section of the California Medical Association at its sixty-seventh Annual Session, Pasadena, May 9-12, 1938.

** To be reported elsewhere.

***Reported in the *Ann. Otol., Rhin. and Laryng.*, *47:* 78-116 (March, 1938), *47:* 402, 411 (June, 1938), *48:* 775 (September, 1938).

the blood constituents may result in unknown chemico-physiological conditions which allow localized stasis or abnormal clotting. But even then, it is likely that the site of the thrombosis is decided by a previous change in the vascular intima. This deduction would attribute all thrombotic and hemorrhagic cerebral lesions to cerebral vascular pathology but a number of other conditions act as very definite factors in their production. All senile individuals must possess sclerotic vessels, yet signs of cerebral involvement do not develop in all of them and in many patients suffering cerebral vascular catastrophes no vascular pathology is demonstrable by present available methods of investigation. Irrespective of this generalization, other factors stand out as direct and indirect attributes in the production of the final process.

One of the unmeasurable, but nevertheless definite contributory factors, particularly in cerebral hemorrhage, is the emotional outburst which not infrequently immediately precedes the ictus. Another hypothetical attribute is interference with the normal innervation of the vascular tree, or in some of its portion at least. It is not reasonable or conceivable that the force of cardiac contractions alone propel and force the entire blood volume content of the body, amounting to approximately one-thirteenth of its weight, throughout the multitudinous branchings of the blood conducting channels. Normally innervated arteries and proper functioning of the mechanisms controlling the reciprocal inter-relationships of pressures in the blood vessels, and in the body cavities as well, must be pre-requisites to normal circulation of the blood.

In this survey of the cerebral vascular lesions found in one thousand individuals, *arteriosclerosis* as a contributory factor in their production was ascribed to the organ most severely affected as demonstrated at the necropsy examination, frequently with the aid of microscopic study of the involved organs. Cerebral arteriosclerosis was considered the principal cause of the cerebral pathology in 43.1 per cent of the cases of cerebral thrombotic lesions and general arteriosclerosis in 10.8 per cent. In cerebral hemorrhage, cerebral arteriosclerosis was the principal causative factor in 19.5 per cent and general arteriosclerosis in 8.1 per cent. Cerebral aneurysms rupturing in twenty patients (4.5%) served as the source of their cerebral hemorrhage.

Forty aneurysms were disclosed in the entire series in thirty-two patients, twenty of them having ruptured with fatal results and twenty aneurysms remaining unruptured in the presence of fatal cerebral hemorrhage from another site. In three patients (0.8%) aneurysms were thrombosed with fatal terminations and in four other patients, who suffered fatal cerebral hemorrhages from ruptured cerebral aneurysms, thrombosed aneurysms were present which had neither ruptured nor been occluded.

Essential hypertension was considered to be the causative factor in 7.6 per cent of the 419 patients suffering fatal cerebral hemorrhage.

Degenerative heart disease was found to be a contributory factor in 16.8 per cent of the thrombotic cerebral lesions and in 21.2 per cent of the cerebral hemorrhages.

Degenerative renal disease was considered the principal contributory attribute in 6.8 per cent of the thrombotic processes and in 8.3 per cent of the hemorrhagic lesions.

Infections served as causative factors more than twice as frequently in the production of cerebral hemorrhage as thromboses, being present in 7.9 per cent of the former and 3.4 per cent of the latter conditions. Cerebral infections, meningitis in nine (2.1%) patients and encephalitis and bulbar poliomyelitis in one each, were present in one-third of the patients in whom infections served as the principal factors in the ensuing cerebral hemorrhages. Another one-third of this group had respiratory infections and the remainder presented varying infections. Various respiratory involvements, gastro-intestinal infections, cellulitis and furunculosis and basal meningitis in one patient, were attributes in ensuing thrombotic lesions, mainly by general depletion of the patient, although low grade infectious involvement of the intima of the implicated cerebral vessel may play a part in both the thrombotic and hemorrhagic processes.

Syphilis was found present in 6.8 per cent of the patients with thrombotic cerebral processes, but there was pathological evidence found in only fourteen (4.0%) of the twenty-four syphilitic patients that syphilis had act-

ed as a direct contributory causative agent in the production of the lethal result. The incidence of syphilis in the patients suffering fatal cerebral hemorrhage was 7.1 per cent, about the same as where thromboses had occurred. In less than one-half this number (3.1%) was there sufficient pathological involvement demonstrated to conclude that syphilis was the direct cause of the hemorrhage present. Ten (2.4%) of this group with cerebral hemorrhage presented syphilitic heart disease and aortitis at necropsy, and meningovascular syphilis was present in three (0.7%).

Chemicals and *drugs* were definite contributory causes of cerebral hemorrhages in eleven patients (2.6%) and of thrombotic lesions in five (1.4%). Morphine addiction had been present in two patients who had lethal cerebral hemorrhages and in two who died following cerebral thromboses. One of the series presented diffuse cerebral thrombotic softening at necropsy following nitrous oxide anesthesia. In one patient each, fatal cerebral hemorrhage followed intraspinous novocaine, application of nupracaine and butyn to the pharynx, intravenous neoarsphenamine, sulpharsphenamin, silver solution treatments in genito-urinary treatments and ingestion of bromides.

Toxemia in the presence of bowel obstruction was followed by cerebral thrombosis in one patient (0.28%) and toxemia of pregnancy was present in seven (1.6%) patients presenting cerebral hemorrhage.

Diabetes was present in eight patients (1.9%) who died from cerebral hemorrhages and a diabetic syndrome was demonstrated in six others (1.4%). Nine (2.5%) of the patients with thrombotic lesions were diabetic and the syndrome was shown in four more (1.1%).

Blood conditions in the form of gastric hemorrhages in two (0.6%) and pernicious anemia in one patient (0.28%) had been followed by thromboses. *Blood dyscrasias* were definitely contributable to fatal hemorrhage in twelve patients (2.8%). Purpura hemorrhagica was the condition in five (1.2%), acute lymphatic leukemia in three (0.7%), chronic lymphatic leukemia in two (0.5%) and pernicious anemia and infantile pseudoleukemia in one (0.28%), each.

Two patients (0.5%) demonstrating pachymeningitis hemorrhagica at necropsy had suffered *head trauma* twenty-seven and forty-two months prior, respectively. In five patients (1.4%) with thrombotic cerebral lesions, surgical procedures with the resultant shock, coupled with the effects of the conditions requiring this form of treatment, were contributory to the lethal result. In one (0.28%) the thrombosis followed fracture of the femur.

Carcinoma contributed indirectly to cerebral thrombotic lesions in four patients (1.12%).

Congenital defects directly contributed to cerebral hemorrhage in five infants (1.2%). The conditions presented were icterus gravis neonatorum in two and the following in one each, congenital heart disease and situs inversus viscerum, congenital absence of the extranepatic ducts and gall-bladder with their adnexia and the thymic status.

Epileptiform convulsions were followed by cerebral hemorrhage in four patients (0.9%) and by thromboses in three (0.84%).

No cause was determined at the necropsy for the hemorrhage present in nineteen patients (4.5%) nor for the thromboses present in nine (2.52%).

Psychoses were present in seventy-three (20.8%) patients of the 351 in whom thrombotic lesions were found and in fifty-nine (11.7%) of the 418 cases of cerebral hemorrhage. The more rapidly fatal termination from the hemorrhagic lesions partially accounted for this variance in incidence but the greater prevalence of cerebral arteriosclerosis (72.6% to 44.0%) in the psychotics with thrombotic lesions was more provocative of the greater frequency of psychotic manifestations in the presence of the more diffuse cerebral involvement. Syphilis was present in three (4.1%) of the seventy-three psychotics with thrombosis and in six (10.2%) of the fifty-nine psychotics suffering fatal cerebral hemorrhage, indicating the attribute of syphilitic endarteritis in the production of both the psychoses and the hemorrhages ensuing. Three psychotic patients (5.1%) suffering fatal hemorrhage presented a diabetic syndrome, one of them hypotension also.

Summary

Infectious processes were demonstrated, in

(Continued to page 238)

Oakwood Sanitarium

A beautifully situated suburban Institution for the modern care of all types of nervous and mental cases.

Cooperative alcoholics and drug addicts accepted.

NED R. SMITH, M.D., *Medical Director,*
Rural Route 6, Tulsa, Oklahoma

SAINT ALBANS SANATORIUM

RADFORD, VIRGINIA

For the diagnosis, care and treatment of Nervous and Mental Diseases and Alcoholism

For rates and further information, address
JAMES P. KING, M.D.

The Colorado Springs Psychopathic Hospital

A Private Hospital for Nervous and Mental Diseases

● Situated in a beautiful valley two miles south of Colorado Springs, which is nationally known as a health center. New building for mild cases of Functional Neurosis, affording complete classification of patients. Home-like surroundings, scientific medical treatment and nursing care. Booklet and rates on application. E. J. Brady, M. D., Superintendent, Colorado Springs, Colorado.

(Continued from page 236)

a survey of 1,000 necropsied cases of spontaneous cerebral vascular lesions, to be most usually provocative of the cerebral embolic and sinus thrombotic processes present, only 6 per cent of the embolic lesions being attributable to mechanical factors.

In the production of thrombotic and hemorrhagic lesions many contributory factors were found to serve as provocative causes common to either type of process. While some agents and processes resulted only in cerebral hemorrhage or thrombosis, others displayed a marked tendency to be followed more frequently by one or the other types of pathology.

Cerebral arteriosclerosis was twice as prevalent in the patients with thrombotic processes as where hemorrhage had occurred. Degenerative heart and kidney diseases were present about equally in the two types of cerebral lesions, as was the incidence of syphilis and epileptiform convulsions. Infectious processes, chemicals and drugs served as contributory factors twice as frequently in the production of hemorrhage as thrombosis while toxemia, particularly toxemias of pregnancy, showed a very high incidence of resultant cerebral hemorrhage in contrast to thrombosis. Blood dyscrasias resulted in hemorrhage over three times as frequently as in thrombosis in this series.

Patients with diabetes showed a greater tendency to suffer thrombotic processes than hemorrhage and those with carcinoma in this survey displayed cerebral thrombosis but none hemorrhage. Cerebral lesions developing postoperatively were much more liable to be thrombotic, but in the presence of congenital systemic defects only hemorrhage was found. Brain tumors were productive only of hemorrhage as was naturally the result in the presence of essential hypertension. Cerebral aneurysms displayed a much greater tendency to rupture than occlude (20 to 3), but among the aneurysms present an equal number had ruptured or had remained unruptured and in four other instances aneurysms present, also in association with cerebral vascular lesions at other sites, had neither ruptured nor been occluded.

Thrombosis occurs in the presence of marked vascular hypertension while hemorrhage may appear where the tension is found

(Continued to page 240)

INCIDENCE OF CONTRIBUTORY FACTORS IN SINUS & VENOUS THROMBOSES (98)

Otitis Media		53
Meningitis		32
Pneumococcic	4	
Tuberculous	1	
Paranasal Sinusitis		5
Cerebral Vascular Lesions		5
Amaurotic Family Idiocy		1
Comedones & Furuncles		14
Dental Infections		2
Tonsillectomy		1
Neck Abscess		1
Respiratory Infections		6
Endocarditis		2
Osteomyelitis		2
Erysipelas		1
Toxemia of Pregnancy		1
Following Swimming		1

ORIGINS OF EMBOLI IN 133 PATIENTS

Cardiovascular 105 (78.9%)		
Cardiac		99
Mitral Vegetations	34	
Aortic Valve Affections	20	
Auricular Thrombi	9	
Ventricular Thrombi	4	
Generalized Endocarditis	6	
Mitral & Aortic Endocarditis	17	
Mitral, Aortic & Tricuspid	6	
Mural Thrombi	3	
Vascular		6
Coronary Thrombosis	1	
Coronary Sclerosis	1	
Aortic Arch Ulcer	1	
Aortitis-Syphilitic	1	
Carotid Artery operative compression	1	
Arteriosclerosis, General	1	
	105	

Extracardiovascular 22 (16%)	
Septic Bronchopneumonia	3
Pulmonary Abscess	2
Pulmonary Infections	2
Paranasal Sinusitis	1
Purulent Otitis Media	1
Appendicitis	1
Pelvic Abscess	1
Fibromyomata, Degenerative	1
Puerpural Sepsis	1
Perinephritis	1
Neck Carbuncle	1
Furunculosis	1
Blood Dyscrasia	1
Cautery—Neck Tumor	1
Cautery—Cheek Tumor	1
Streptococcic Sore Throat	1
Oesophageal Ulcer	1
	22
Source not determined at Necropsy	6
	28

GLENSIDE

JAMAICA PLAIN, BOSTON, MASS.

• A small, attractively located sanitarium for nervous, mild mental or chronic illnesses. •

MABEL D. ORDWAY, M.D.

6 Parley Vale Tel. Jamaica 0044

PASADENA SANITARIUM

(Founded 1906 by T. W. BISHOP, M.D., 1870-1938)

INTELLIGENT AND UNDERSTANDING CARE FOR ALL TYPES OF NERVOUS AND MENTAL CASES.
SELECTED CASES OF ALCOHOLISM AND HABITUATION.

Located on a secluded 33 acre tract—Seven miles from downtown Los Angeles

G. H. REINHARDT, M.D.
Medical Director

T. W. BISHOP, JR.
Executive Director

— P. O. Box 111 — South Pasadena, California —

Tucker Hospital

212 West Franklin Street (Corner Madison)
RICHMOND, VIRGINIA

A private hospital for the study and treatment of Neuropsychiatric and Endocrinological Cases

Under the Charge of

Drs. Beverley R. Tucker, Howard R. Masters, and Jas. Asa Shield

THE HARDING SANITARIUM

WORTHINGTON, OHIO

For Nervous and Mental Disorders

NINE MILES NORTH OF STATE HOUSE—COLUMBUS

GEORGE T. HARDING, M.D., *Medical Director*
HARRISON EVANS, M.D.

FRED H. WEBER, M.D.
MARY J. WEBER, M.D.

•

SUBSCRIPTION PRICE

Diseases of the Nervous System

— $2.50 ANNUALLY —

•

(Continued from page 238)

subnormal, the *relative* changes in pressure requiring consideration as contributory mechanisms in the production of the lesions.

It seems quite questionable whether thrombosis or hemorrhage occurs without local pathological involvement of the vessel, but other conditions act as very definite factors in their production. Chronic debilitating diseases are prone to be followed by thrombotic processes and infections; chemicals and other exogenous factors are generally more productive of the hemorrhagic lesions.

Indeterminate contributory factors are emotional reactions and interference with normal innervation of the vascular tree as well as proper reciprocal pressure relationships in the body cavities, channels and vessels.

1930 Wilshire Boulevard.

CONTRIBUTORY FACTORS IN CEREBRAL THROMBOSIS & HEMORRHAGE

	Thrombus (351)		*Hemorrhage* (418)
	Exclusive Sinus Thrombosis (98)		
	%	%	%
1. Arteriosclerosis		54.7	27.6
Cerebral	43.1	19.5	
General	10.8	8.1	
2. Aneurysm (Thrombosed or Ruptured)		0.8	4.5
3. Brain Tumor			0.7
4. Degenerative Heart Disease		16.8	21.2
5. Essential Hypertension			7.6
6. Degenerative Renal Disease		6.8	8.3
7. Infections		3.4	7.9
8. Syphilis		6.8	7.1
9. Chemicals		1.4	2 6
10. Toxemia		0.28	1.6
11. Diabetes		2.5	1.9
12. Blood Dyscrasias		0.84	2.8
13. Post Operative & Trauma		1.7	0.5
14. Carcinoma		1.12	
15. Congenital Defects			1.2
16. Epileptiform Convulsions		0.84	0.9
17. No Cause Determined		2.52	4.5
		100.00	100.00
Psychoses Present		20.8	11.7

ARE YOU INTERESTED
IN
"VOICE DISORDERS AND
PERSONALITY CONFLICTS!"

We are publishing an article under this title in the July issue of

DISEASES OF THE EYE, EAR, NOSE AND THROAT

A new and practical journal edited by

Francis L. Lederer, M.D.,
Chicago, Illinois.

Samuel J. Meyer, M.D.
Chicago, Illinois

Write in today for your **free** sample copy.

NEXT MONTH WE PRESENT

DEPRESSIONS
Wm. C. Menninger, M.D., Topeka, Kansas

WORK OF THE RECORDERS COURT CLINIC IN DETROIT
Lowell S. Selling, M.D., Detroit, Michigan

THE DOCTOR AND HIS NERVOUS PATIENT
Mary G. Schroeder, M.D., Chicago, Illinois

JITTERBUG AGE
W. C. McConnell, M.D., St. Petersburg, Florida

SOME THOUGHTS ON THE PSYCHOLOGY OF POST-OPERATIVE CONVALESCENCE
Florence Clothier, M.D., Boston, Massachusetts